

VOLUME 9

New York Number

NUMBER 2

THE
MEDICAL CLINICS
OF
NORTH AMERICA

SEPTEMBER, 1925

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR) BY W. B. SAUNDERS COMPANY, WEST WASHINGTON
SQUARE, PHILADELPHIA

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THE MEDICAL CLINICS OF NORTH AMERICA

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CONTRIBUTION BY DRS RUSSELL L CECIL AND
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PHYSICAL THERAPY IN CHRONIC ARTHRITIS: ITS USES AND LIMITATIONS

In any discussion of the treatment of chronic arthritis a preliminary definition of terminology is necessary. Various classifications of arthritis have been described, based on pathologic, etiologic, or clinical distinctions. Perhaps the most satisfactory classification of this disease is that of Nicholls and Richardson, which separates all infections of the joints into two main divisions: (1) Proliferative arthritis, and (2) degenerative arthritis. In proliferative arthritis the changes are of a frankly inflammatory nature. Granulation tissue forms between the articular surfaces with ultimate injury to the cartilage and bone and the production of fibrous or bony ankylosis. In addition to these changes there is more or less inflammation of the periarticular structures which may result in thickening about the joint and, with shrinkage of the new fibrous tissue, in deforming contractures. In the early stages this type of arthritis may often be subdivided into cases that affect primarily the joint itself and those in which the inflammation is periarticular. In either of these subgroups, however, the progress of the disease may eventually lead to a mixed form in which both the joint and the periarticular structures are involved. Infectious arthritis, arthritis deformans, and the various specific arthritides fall into the proliferative group.

NOTE.—The next number of the MEDICAL CLINICS OF NORTH AMERICA will be a continuation of the New York number.

S. M. S. Medical College

Degenerative arthritis sometimes called hypertrophic, is characterized primarily by a new growth of bone in and around the articular surfaces. In this type true ankylosis does not occur, but the "lipping" of the articular surfaces may eventually lead to more or less locking of the joint. Heberden's nodes, morbus coxæ senilis, the arthritis of menopause, and senile arthritis all fall in this group. As the name implies, the lesions in these cases are more degenerative than inflammatory and are probably not referable to infection.

A rational discussion of the use of physical therapy in arthritis must also take account of the *stage* of the process. We must distinguish between cases that are in the acute or progressive stage, and those in which the process has become stationary. A third group would include cases which already show signs of spontaneous recession. These groups might be designated the progressive, stationary and receding types.

In the third place, the physician must make every effort to determine the *etiology* of every case of arthritis. This should be done before any form of therapy is undertaken. Foci of infection are responsible for a large percentage of the cases. On the other hand, other factors are probably at work in the degenerative forms. For example, menopause arthritis, quite a common type, is rarely associated with any demonstrable focus of infection.

From these preliminary considerations it is clear that the main essentials in the successful treatment of arthritis are a thorough comprehension of the morbid anatomy of the disease and the determination of the actual stage of the process in any given case. It is equally obvious that no method of treating arthritis can be permanently successful unless the *etiology* is determined and the exciting cause removed.

THE MODERN TREATMENT OF ARTHRITIS

At the present time the recognized treatment of chronic arthritis consists of (1) Removal of infectious foci and other toxic factors, such as intestinal intoxication. (2) Medicinal treatment consisting of salicylates, iodide, thyroid extract, etc.

In administering these agents the physician has several objects in view—the control of pain, softening of fibrous tissue, stimulation of metabolism, and other less clearly recognized effects (3) Specific treatment with vaccines as, for example, gonococcus vaccine injections in gonococcus arthritis, streptococcus vaccine in cases associated with streptococcus tonsillitis, etc (4) Non-specific therapy Administration of proteins such as milk and typhoid vaccine for the purpose of producing non-specific reactions (5) Surgical treatment This consists, in the more acute cases, in immobilization with splints, and traction to prevent adhesions and ankylosis In chronic cases the functions of the orthopedic surgeon are to prevent contractures and break up ankylosed joints by arthroplasty or forcible rupture of adhesions under anesthesia (6) Physical therapy The various forms of physical therapy each have their definite functions, depending upon the kind of physiologic reaction each gives rise to Some of these agents are employed to produce a general, others to produce a local, effect Arthritis being a systemic disease, the treatment should be directed not only toward the local manifestations in the joints, but toward correction and improvement of the patient's general condition

In the present discussion we will confine ourselves to a consideration of the last-named therapeutic measure Physical therapy is coming into greater and greater popularity, and deservedly so But as in the case of all new agents, we are in danger of being carried away by our enthusiasm, and of applying physical therapy to cases where it is not indicated, or expecting results beyond those which physiotherapy is capable of achieving

Before taking up the treatment of the various forms of chronic arthritis it would seem desirable to review briefly the various forms of physical therapy now in use, considering under each heading the physical qualities of the agent employed and the character of the physiologic reaction which it arouses in the tissue

The agents now most frequently employed are

- 1 Heliotherapy
- 2 Heat

3. Electricity
4. Radium and γ -ray
5. Hydrotherapy —
6. Massage
7. Mechanotherapy and exercises

These various forms of physical therapy will now be considered in detail

Heliotherapy.—Finsen, of Denmark, was probably the first in modern times to apply sun treatment scientifically. He started his work in 1890 and confined it mainly to the treatment of lupus. Dr. Rollier, in Leysin, Switzerland, opened a sanitarium in 1903 for the treatment of extrapulmonary tuberculosis with heliotherapy and was followed by Calvé and Jaubert and others in France, and in this country similar work was started in Denver and Buffalo. By gradual steps our knowledge of the therapeutic value of the sun rays has been developed, although there is still much to be done.

In studying the sun spectrum it is convenient to divide it into

1. Actinic or ultraviolet rays
2. Luminous or light rays
3. Caloric or heat rays

Of these, the caloric rays are found among the least refracted rays of the spectrum and have a longer wave-length. The ultraviolet rays are most refracted and have a short wave-length, and the luminous rays are found between the two. These rays are not three well-defined regions of the spectrum but overlap widely. The Finsen school emphasizes the whole sun spectrum while Rollier ascribes his results to the ultraviolet rays and the altitude. The application of the sun being impractical in large cities, attention was focussed on the possibility of employing some form of artificial sunlight. The result has been that we have lamps for heat rays, for light rays, and for the ultraviolet rays, but no lamp for the whole spectrum. Accurate knowledge of the physiologic effect of these rays is much behind their practical application, but of late much work has been done both in this country and abroad on the physiology of the ultraviolet rays.

There are several different types of lamps for the ultraviolet rays. Those used by the authors are mercury vapor lamps. The ultraviolet rays are produced by leading slow tension direct current through two mercury electrodes contained in a quartz burner.

The ultraviolet rays are invisible. The rays of the spectrum are measured in wave-lengths and the unit is called an Angstrom unit. The physiologic effects of ultraviolet light are

1 *Bactericidal Effect*—The bactericidal effect of the ultraviolet rays is well known from their use in sterilizing drinking-water and swimming pools. Paccini of Chicago, has determined the death points of various bacteria suspended in clear solutions in direct contact with ultraviolet light. It varies from six to twenty-five seconds for the common pathogenic micro-organisms.

2 *Ultraviolet Causes an Increase in the Calcium and Phosphorus of the Blood-plasma*—The benefit of ultraviolet rays in rickets has long been known and also the benefit of cod-liver oil. It was only recently that Kugelmass and McQuarrie demonstrated the fact that cod-liver oil when oxidized gives off ultraviolet rays. It has also been proved that calcium medication in combination with ultraviolet radiation will increase the calcium content of the blood-plasma.

3 *Leukocytosis, General and Local*—Both the general and local application of ultraviolet rays is followed by an increase in the leukocyte count. Whether this is due to increased production of white cells or only a leukocytosis in the peripheral circulation in response to irritation it is difficult to say.

4 *Pigmentation*—It was formerly believed that the amount of tanning which followed ultraviolet radiation was an index of its beneficial action. The pigmentation is considered by some a defense reaction of the skin. This has been disputed of late and the similarity of the hemoglobin in the blood to the chlorophyll in plants has been advanced but not experimentally proved.

5 *Metabolic Changes*—The clinical results are not yet explained physiologically, but probably depend on the reaction of the different amino-acids to the ultraviolet rays and also on

the influence of ultraviolet rays on the nucleus and cytoplasm of the cellular structures

6 *Counterirritation*—The frequently observed relief in obscure nerve pains such as those of neuritis and neuralgia is probably due to the counterirritant action of ultraviolet rays

Practical Application of Ultraviolet Rays—For practical purposes two ultraviolet lamps are available

1 The air-cooled lamp, which gives mainly the long waves. The chemical action of the long waves is chiefly an oxidizing one, hence they tend to increase metabolism

2 The water-cooled lamp, which gives the short waves. The latter have a decidedly bactericidal action. Chemically they act as a reducing agent and depress metabolism

The air-cooled lamp is used mainly for general radiation of the entire body. Its action is mostly on the blood. In order to have the maximum of blood radiated it is customary to produce a general hyperemia of the skin by means of a short exposure (ten minutes) to a radiant light

The exposure to the air-cooled lamp should start with three minutes at a distance of 60 to 70 cm. and the time increased until a first-degree erythema results. In calculating the dosage we have to consider the amount of pigment in the patient's skin. The Nordic type stands much less radiation than the dark-skinned type from South Europe

The water-cooled or Krommayer lamp is used mainly for local application. When used in direct contact with the skin we start with fifteen seconds and increase with fifteen seconds each treatment. Each lamp has to be standardized by itself and this ought to be repeated at least once a month because the lamps deteriorate somewhat. Under certain conditions, when both a general and local action is desired, it is advantageous to combine the air-cooled and the water-cooled rays

Heat—When heat in any form strikes the human body an increased surface temperature is the first noticeable change. In order to protect the body the heat-regulating mechanism responds with a hyperemia; the capillary bed is engorged with blood and thereby congestion somewhere else is relieved. Other

avenues of heat dissipation are utilized to equalize the temperature, such as increased pulmonary ventilation and evaporation through the sweat glands. The reaction of the skin to heat and sweat becomes less acid or more alkaline. The action of heat plus hyperemia on the peripheral nerves is sedative and by the reflex arc this sedative action is admitted to deeper structures.

The practical application of heat may be general or local. Its general application is described under hydrotherapy. In local application of heat the following means are available:

1. The arc light, which is best employed by the ordinary marine search light with the glass front removed. The light is thrown in parallel rays by a parabolic reflector. The light value is about 5000 candle-power. These arc lights usually go under the name of radiators.

2. The high power incandescent lamp has a tungsten filament of 500 candle-power and is provided with a dome reflector. The carbon filament gives off more rays while the tungsten filament lamp produces a smaller amount of luminous rays with very little heat.

3. Baking and bakers are passing out of vogue, and heaters should be substituted for any box or heater. Heat is produced in the same way as in an electric lamp by carbon filaments. The cleanest application is a battery of 8 to 36 carbon lamps. The part treated should be covered by a blanket. The time of application is ten to fifteen minutes.

Various forms of heat are usually applied as preliminary measures for electrical treatments, massage, or muscle exercises.

Electricity.—The hesitancy which many medical men are advocating electricity as a therapeutic agent is due to the excessive claims of the electrotherapeutists. They often base their work on a knowledge of electricity rather than upon a knowledge of general medicine, and are electricians practising medicine rather than medical men using electricity. During recent years especially since the last war much has been written for and against the use of the electric

currents We will try here to take up the action of these currents as based on our knowledge of physiology and pathology

1 *Galvanism* —The unmodified galvanic current is a direct, continuous, unidirectional current, usually obtained from dry cell batteries or from the street current with the interposition of resistance. It is applied in two ways, continuous galvanism and interrupted galvanism

Continuous Galvanism —This is applied by means of two metal electrodes usually of the same size, with a pad or several layers of gauze soaked in normal saline or some other appropriate solutions. These electrodes are connected to the machine, one becoming the positive, the other one the negative electrode, with positive and negative polarity changes. Great efforts have been made trying to explain what takes place in the interpolar path through the tissues of the body. The result is the medical ionization theory which is based on what we know of electrolysis or ionic interchange. Many enthusiasts believe that by this method it is possible to introduce medicine through the skin and even through a joint. Experiments have not proved this, but it seems rational to believe that the tissue resistance will produce some heat and thereby benefit, at least temporarily, such conditions as sciatica. The softening effect around the cathode is made use of to loosen up scar tissue.

Interrupted Galvanism —If a large electrode is applied over the brachial plexus and a small hand electrode over the motor point of the biceps muscle on the same side and these electrodes connected with a galvanic battery, we will obtain one contraction of the biceps at the make and one at the break of the current. These contractions are rapid and vigorous. Even if the musculocutaneous nerve is cut there will still be a contraction at the make and one at the break, but now the contractions are sluggish. From this we conclude that the galvanic interrupted current stimulates through both the nerve end-point and the muscle-fibers. Interrupted galvanic current therefore suggests itself for muscle exercising in cases where muscle atrophy is due to degeneration of the nerve or of the muscular tissue.

2 *Faradism* —This is the alternating current obtained from

an induction machine This consists of a galvanic current leading through an interrupter into a primary coil which is in induced relation to a secondary coil This current can be modified as to number of interruptions, voltage, and amperage The faradic current is applied very much like the interrupted galvanic current with one indifferent electrode over some part of the spine and the small hand electrode over the motor point of a muscle The contraction that results is a consistent, powerful contraction of the muscle

If this kind of current is applied to a muscle without nerve supply, we get no response From this we conclude (1) That the faradic current stimulates only through the nerve ending (2) That the faradic current is useful in diagnosing peripheral nerve lesions

3 *Sinusoidal Current*—We will confine our discussion to the slow sinusoidal current which is the straight galvanic current, modified by a varying resistance into symmetric waves with alternating polarity

By reason of this reversing polarity of each successive wave the sinusoidal current gives no polarity effects It provokes a powerful contraction, nearly painless, because maximum strength of current is gradually approached and gradually receded from Because of the gradual contraction and gradual relaxation of the slow sinusoidal and its painless application it has a wide field of usefulness

In the interrupted galvanic, the faradic, and slow sinusoidal currents we have the mechanical agents for producing muscular contractions These contractions cause a temporary increase of the circulation and therefore of the nutrition to the muscle, and when repeated a permanent increase in volume strength, and tonicity

The technic is not easy and requires a knowledge of anatomy and physiology and experience in the application of electricity It is necessary to know what muscles are to be treated and to use only enough current to contract these muscles and not their antagonists There is danger of overworking weak muscles and thereby doing more harm than good

There are two methods of applying the sinusoidal current the unipolar and the bipolar method. In the unipolar method a large electrode is placed over the upper part of the trunk or the brachial plexus in treating upper extremities over the lower spine or lumbosacral plexus when the lower extremities are concerned, and the small hand electrode is applied over the motor point of a muscle. In the bipolar method we use two small electrodes, one at either extremity of the muscle.

It is dangerous to apply these currents in the neighborhood of a heart which responds with fibrillation when stimulated. Neoplasms and acute infections are also contraindications. These electric stimulations by no means replace the so-called Swedish movements translated by Lovett to muscle training and wherever it is possible to contract a muscle by voluntary impulses no effort should be made to replace the normal brain impulse by an electric current.

4 *High frequency Currents*.—In therapeutics the dividing line between high and low frequency currents is at the point where human tissues cease to respond to each impulse. This point is above 30,000 oscillations per second. If the voltage is raised and the frequency increased above 30,000 oscillations we have the high-frequency current of d'Arsonval or the diathermy current. If the voltage is stepped up a second time by means of a Tesla coil or an Oudin resonator we have the Tesla current and the Oudin current.

In the d'Arsonval current the voltage may be so high that in spite of the high frequency of oscillations sensations and even contractions may result. In the diathermy current the voltage is so much lower and the frequency so much higher that no sensation is experienced except heat.

The most important high-frequency current is diathermy which means heating through and through. The diathermy machine is a high frequency machine producing a relatively low voltage and high average current with one to two million oscillations per second. An electric current passing through a conductor develops heat depending on the resistance of the conductor and the intensity of the current. The diathermy cur-

rent produces heat in the tissue traversed by it as it passes between the electrodes. This heat production is the only effect, there is no contraction, shock, or electrolytic changes as far as we know.

To Dr. Nagleschmidt, of Berlin, goes the credit of working out a machine capable of producing such a current. During the late war the machine was further improved in this country and today it is one of our most useful therapeutic agents.

Surgical diathermy or electrocoagulation is simply a cooking process obtained when a large electrode and one of pin-point size are used. The heat at the small electrode is sufficient to coagulate tissue cells. This is not to be confused with the actual cautery where burning or incineration of the tissues takes place.

Medical diathermy may be either stimulative or sedative. In medical diathermy two electrodes, usually of the same size, are used. The dosage is calculated from the size of the electrodes used and the resistance of tissue traversed, but the best indication is the tolerance of the patient.

In the *stimulative* technic the current is turned on suddenly up to about 700 m amp for six to seven minutes and is turned off suddenly. Thereby no great heat is developed and the object is to irritate the tissue between the electrodes to greater activity.

Absorptive or *sedative* diathermy. To produce this effect a higher amperage is necessary, sometimes up to 2000 m amp or more for a period of fifteen to forty-five minutes. The current is turned on slowly until maximum amperage is reached. This raising of the amperage should take three to five minutes. The treatment is likewise terminated by turning off the current gradually. The immediate effect is increased heat production and the heat-regulating mechanism of the body responds with a local hyperemia. With this local hyperemia follows an increase of the blood constituents, including the leukocytes. Because of the increased blood-supply stasis is broken up and deposits are made soluble. The action of heat and hyperemia on the sensory nerves is sedative and, therefore, the diathermy current is one of the best anodynes we have.

Oudin—This is a high-frequency current connected to a vacuum or non-vacuum electrode, which acts as a condenser electrode. This is sometimes called indirect diathermy. It is used with the electrode in direct contact with the skin when a sedative form of heat is desired. A counterirritant effect is obtained by holding the electrode a short distance from the skin. A shower of sparks or an effluve will then pass from electrode to skin.

Autocondensation—This is another application of the high-frequency current. The patient holds in his hands an electrode in the form of a metal rod, which is connected with one terminal of the high-frequency current. The patient lies on a special table or pad connected with the other terminal. This autocondensation pad consists of a metal plate with insulated covering. The pad electrode, with the patient and the intervening insulator, make up an apparatus not unlike a Leyden jar. The whole body heats up gradually and complete relaxation follows. If continued long enough, profuse perspiration occurs. The treatment takes about forty-five minutes and has a reputation for lowering the blood-pressure. If by autocondensation we could markedly increase the body temperature we might have a substitute for the foreign protein reaction, now much used in therapeutics.

5 *Static Machine*—The static machine is a self-charging condenser machine with a very high voltage and comparatively small amperage. The currents obtained from such a machine have been used empirically for many years. In the hands of men who understand the technic of the static machine it is a most valuable factor in therapeutics. It has been used without judgment by many unfamiliar with the technic, until the medical profession today shows more prejudice against the static machine than against any other form of electricity. The principle involved in the static machine is easily understood.

The static machine is a self-charging condenser of several thousand volts which can be discharged with or without interruptions to a patient, either generally or locally, in a concentrated spark or in a diffuse breeze. The machine consists of

eight or more plates which rotate against the friction of brushes. Positive and negative electricity is collected at combs which are connected with two discharging rods or terminals. The capacity of the machine is increased by Leyden jars. According to the hook-up and the different methods of discharge, we speak of *static spark*, *static wave*, *static effluve*, *static induced*, and *static saturation*.

Static Spark—The patient sits on an insulated platform, connected with the negative pole. No Leyden jars are used. The spark ball is grounded. In the direct static spark the positive pole is grounded and the sparks are administered directly from the machine, the whole capacity of which is concentrated in the spark. In the indirect spark the positive pole is not grounded. Sparks are drawn off the patient by the spark ball, which is an electrode with insulated handle, terminating in a metal knob connected with a grounding chain.

The physiologic effect of the static spark is a sudden contraction of all contractile tissue under the spark. These contractions are followed by an increased circulation and metabolism in the parts treated.

Static Waves (Condenser Discharge or Morton Wave)—A metal plate is attached to that part of the patient to be treated and then connected with the positive pole. Leyden jars are connected up and the negative pole is grounded. At the beginning of the treatment discharging rods are closed and opened to regulate the speed and discharge of the wave.

This is the most valuable of the currents obtained from the static machine. While the static spark is a painful procedure, the wave is easily endured by anyone. Here the discharges come in waves and the result is a repeated mechanical contraction of all contractile tissues underneath the plate electrode. It is the best means we have to relieve the swollen tissues following a sprain and replaces massage in myositis and prostatitis. It is also useful in chronic arthritis as a local treatment when the lesions are parieticular.

Static Effluve—The same hook-up is used as in the direct spark. The discharge is diffused through a dry stick or any

material with high electric resistance such as the DeKraft pencil. The effect is the same as that of the spark except that the discharge is much diffused and can be used over any delicate part.

Static Induced —The patient needs no insulated platform, as this is a bipolar application. One electrode from the patient is connected with the outside coating of a Leyden jar, the other electrode from the patient to outside of the Leyden jar. The Leyden jars are not connected directly, and neither side of the static machine is grounded. The main terminals are closed at the start of the treatment and are then opened up until the tolerance of the patient is reached. The effect is a powerful compression of tissue between the electrodes.

Static Saturation —The patient sits on an insulated chair and is charged to full saturation for ten to twenty minutes. The physiologic effect is not well understood, but the psychologic effect is often striking.

Radium and x-Ray —The local effect of radium and x-rays seems to be very much the same, namely, the production of fibrosis, an action which theoretically, at least, would not appear to be of much value in the treatment of arthritis. It is a well-known fact that many patients with arthritis undergo radium or x-ray treatment for neoplasms without any subsequent improvement in the arthritic condition.

It is possible, of course, that minute doses of radium or x-ray, by stimulating the lymphocytes, might affect an arthritic joint favorably by increasing local phagocytosis.

A considerable amount of the benefit which patients receive at various spas is now attributed to the radio-activity of the water which they drink or bathe in. Just how much basis there is for this belief it is hard to say.

The soluble salts of radium are now employed intravenously as well as by mouth and for bathing purposes. It is claimed that radium promotes secretion, excretion, and the metabolic processes, and favors the elimination of nitrogen. Experimental evidence, however, in favor of these claims is still meager.

Hydrotherapy —The practical application of water in ther-

apeutics involves two factors, the physiology of the skin and the physical characteristics of water

The body covering consists of epidermis for the protection of the body and a mesodermal layer called corium

As protective agencies for the skin there are

- 1 The external stratum corneum of the epidermis, the peripheral cells of which consist of keratin, a highly resistant substance and insoluble in mineral acids

- 2 The secretion from the sebaceous glands, consisting of fats, soaps, etc., which make the skin a waterproof covering for the body

- 3 The pigment granules present in the stratum mucosum may also be considered as a protective agent against luminous and actinic rays

The part that the skin plays in the heat-regulating mechanism is an important one. The factors concerned are the blood-supply to the skin, the sweat glands, the muscular fibers, and the cutaneous nerves. The papillæ which project into the epidermis contains loops of minute blood-vessels, arterial and venous capillaries, which together with the lymphatics nourish the skin and effect the exchange of gases and the secretions of the sweat glands

The sweat glands are numerous small glands situated in the subcutaneous tissue with ducts ending on the surface of the epidermis. The sweat glands contain secretory nerve-fibers. In ordinary life the usual cause for profuse sweating is an external high temperature or muscular exercises. The high temperature acts on the cutaneous sensory nerves which, in turn, reflexly stimulate hyperemia and perspiration. This reflex response constitutes a very important means of regulating the body temperature

The cutaneous muscular fibers are involuntary muscles which also help to regulate secretion and circulation. The cutaneous nerves supply the skin with sensation. Sensory impulses of all kinds are conveyed to vasomotor, respiratory, heat-regulating, cardiac, and other centers in the medulla, which respond as the occasion demands to protect the organism from injurious external influences

The Physical Properties of Water—Water occurs in three physical forms—solid, liquid, and vapor—which only means different temperatures. These modifications of water make it a most useful agent in therapeutics for conveying heat or cold to the body.

Water being a good conductor of heat and cold, it absorbs and gives off heat and cold very readily in moderate humidity. If we stand nude in a bathroom at a temperature of 80°F , then submerge in a tub of water at the same temperature the difference in sensation before and after submersion is quite marked. The chilly sensation after such a bath is due to the decrease in body temperature caused by the evaporation of water from the skin. It is said that water gives off its temperature twenty-seven times more rapidly than air.

The mechanical impact of water delivered under pressure on the skin acts very much like massage or the static wave current.

Much of the ancient application of hydrotherapy and of the present medicinal baths cannot be defended by our present knowledge of physiology. The claims that carbon dioxide, sulphur, or radio-activity are absorbed through the skin are questionable and the use of medicinal baths, both natural and artificial, is empirical to say the least.

When warm water is applied to the skin it induces a hyperemia of the skin, increased activity of the sweat glands, relaxation of the cutaneous smooth muscles, deeper respiration, and a sedative effect on the nervous system.

In the application of cold water we have blanching of the skin or withdrawal of the cutaneous circulation, decrease in the activity of the sweat glands, contraction of the cutaneous muscles and a general tendency to activity which may produce shivering to make up for the loss of heat.

For practical purposes we may say that water below the body temperature down to 40°F stimulates and the water above body temperature up to 115°F acts as a sedative. These statements are based on subjective symptoms of normal individuals but the objective symptoms correspond very closely to the patient's sensations, and from the action of water in health

we may draw conclusions about the action of water in disease

The Methods of Applying Hydrotherapy—These methods of applying hydrotherapy have undergone considerable change during the last twenty years. The different forms of packs, although very beneficial, are seldom seen in general hospitals today. Few doctors know when and how to prescribe them and few nurses are familiar with the technic of administering them.

1 *The Douche*—A jet or shower of water under pressure. The initial temperature of the water is 100° to 115° F. and this is gradually decreased to 90° F. or less after two or more minutes. The end-result should be a "reaction" which is nothing more than a general stimulation of body functions.

The *Scotch douche* is applied by two jets differing 40° to 50° F. in temperature with the control table 12 to 14 feet from the patient. It is applied mainly over the muscular part of the body, i. e., the back and extremities. It is a powerful stimulant, a form of circulatory gymnastics. It can be modified by the operator's finger into a fan douche and applied to any part of the body.

2 *General Immersion Baths*—This type is much used in neurologic and psychiatric cases because of the resulting muscular relaxation and sedative action. The temperature should be 2° to 5° F. above body temperature. Its duration depends on the particular case. It is often combined with massage and manipulations under water.

3 *Vapor Baths*—The limit of toleration of the body for moist heat is about 130° F. This application produces a marked elimination and removes epidermal accumulations.

Vapor baths at lower temperature are soothing and sedative. Vapor baths that produce profuse sweating should be followed by the application of cold water.

Electric Cabinet Baths—These are built either as vertical boxes where the patient sits on a chair with the head through an aperture or the patient reclines in a horizontal box with the head outside. The box contains a number of light bulbs which are switched on from the outside. Where elimination is desired

the Mazda tung-ten filament bulbs are used. Towels soaked in cold water are applied to the head and cold water is given to the patient to drink. This treatment takes from five to twenty minutes.

The cabinet bath is followed by the Scotch or fan douche. The patient is then well dried and moderate exercise advised. Dr. Holder, after experiments, summarized the changes after fifteen minutes' exposure in an electric cabinet bath with Mazda lamps as follows:

1. General rise of temperature, pulse, and respiration
2. No demonstrable change in hemoglobin
3. No demonstrable change in red cell count.
4. Diminution of white cell count
5. Differential count was unaltered

Local Application of Hydrotherapy—1 *Wet Compresses*—These are very popular and consist of two or more folds of old linen wrung out of either warm or cold water at desired temperature and covered with flannel. They may be applied to any part of the body.

2 *Whirlpool baths* are used for the extremities. They consist of a basin preferably of Monel metal, shaped either for the upper or for the lower extremity. Water at 95° to 110° F. is forced into this bath under pressure and at an angle to form a whirl. The outlet pipes are usually constructed with an aeration tube by which air is sucked in with the water. It is often advisable to have two or four inlet pipes to produce a real whirlpool. The benefit from such a whirlpool bath comes from the cleansing properties of the water, the temperature, and the massage effect of the whirl.

3 The *contrast bath* is simply two basins of water at different temperatures. One contains water of 100° to 115° F., the other vessel has cold water with ice floating in it. The limb to be treated is first immersed in the warm water for ten minutes, then plunged into the cold water and kept there for half a minute. It is a very powerful vasomotor stimulant.

The Prescription of Hydrotherapy.—In order to obtain the best results, the hydrotherapy prescription should specify the

temperature, duration, and pressure of the water to be used and careful observations should be made after each treatment. The method of administration should not be left entirely to the judgment of a trained technician.

High colonic irrigations are really a form of hydrotherapy. Of late years they have come into great vogue. They are undoubtedly valuable in various forms of intestinal stasis and intoxication, but if kept up too long they may induce an atony of the intestinal muscles.

The relation of intestinal infection and intoxication to arthritis is difficult to determine. In cases where definite colitis exists an associated arthritis may, with reason, be attributed to the intestinal lesion. On the other hand, many cases of arthritis are constipated or flatulent, and the physician who regularly attributes these infections to the intestinal disorder will usually miss the real cause of the disease, which in the infectious cases will usually be found in the tonsils or teeth, and in the degenerative cases cannot be attributed to infectious focus of any kind.

It must be admitted, however, that while arthritis is rarely cured by colonic irrigations alone, they undoubtedly help to build up arthritic patients who are in a low state of health, and for this reason are often indicated in middle-aged women who are constipated and much over weight. Patients of this type are usually suffering from menopause arthritis, but infectious arthritis also occurs among them with a moderate degree of frequency.

Massage—The action of massage is twofold: mechanical and reflex.

The mechanical action of massage may be exerted in a number of ways: (1) By stimulating the circulation, (2) by aiding the movement of lymph, (3) by tension on structures which it is desirable to free or stretch, (4) by pressure on abdominal viscera.

1. The *circulation is stimulated* by assisting venous return and by restoring the tone of the vasomotor system. By assisting the venous return the column of blood in front of the arterioles

is lightened and the blood can then pass through them more rapidly. Assuming that the blood-pressure is constant a larger supply of blood will be required for the part under treatment, and the vasomotor mechanism must be called into play to fulfil this want. The healthy glow of the skin often seen after massage is due to a dilatation of the arterioles as a result of their temporary paralysis.

In a normal body muscular contraction serves as a pump on the venous flow. Massage should aid the venous return in the same way. Such pumping of the venous blood is feasible only when the lumen of the vein is open, complete muscular relaxation is therefore essential.

2 *Effect of Massage on Lymphatics*—If massage aids the venous return, it also indirectly accelerates the flow of lymph. Since pressure in the lymphatics is very low, only the lightest massage is necessary. Stagnation of lymph or edema is temporarily reduced by elevation, but this does not restore the tone of the vasomotor system. Elevation or, better, vasomotor exercises should be combined with massage to insure permanent relief.

For the reason that the stopper must be removed before emptying a bottle, manipulations must be started above the edema before any local treatment can be effective.

3 *Mechanical Effect of Massage in Stretching Tissue*—Restoration of function is often prevented by pathologic processes which result in a new growth of connective tissue. Forceful stretching is required to break up fibrous tissue and this should be done only under anesthesia. It is in the yellow, elastic adhesions that massage and exercises are indicated for in most instances such adhesions gradually yield to this form of treatment.

4 *Massage on Abdominal Viscera*—Much has been written, but nothing proved regarding the effect of massage on the secreting glands in the gastrointestinal tract. There is no doubt, however, that the colon can be emptied by massage, which is also used to great advantage in cases of visceroptosis, owing to its combined mechanical and reflex action.

The reflex action of massage has been questioned, but it is only reasonable to suppose that if one form of surface stimulation can produce a muscular contraction by reflex action, another form of stimulation can secure relaxation. Perhaps the most convincing proof is the experience of Dr. Lucas Champomiere, who treats recent fractures with gentle massage and secures relief of spasm, pain, and swelling, so that reduction of the fracture is obtained with greater ease.

Massage differs radically from active exercises in its capacity to modify the condition of the muscular tissue without fatiguing the patient or even employing his will power.

According to Pemberton, massage of voluntary muscles is not accompanied by evidences of lactic acid production and acidosis that attend relatively mild exercises. Nor does it bring about the loss of acid and the alkalosis that characterizes therapeutic exposure of the body to external heat.

Krogh and his school are of the opinion that the benefits of massage are due to changes in the blood-supply, especially the capillary circulation.

Champomiere has demonstrated the benefit of massage on muscular tissue by crushing such tissue and applying massage to some parts and not to others. The tissue which was not massaged showed a dissociation of the muscular fibers into fibrillæ as shown by well-marked longitudinal striation, hyperplasia or simple thickening of the connective tissue and increase in numbers of the nuclei, interstitial hemorrhages, and enlargement of the blood-vessels with hyperplasia of their adventitious coats. The sarcolemma showed a multiplication of nuclei resembling interstitial myositis.

In the massaged tissue the muscle appeared normal, secondary fibrous bands separated the muscle-fibers, there was no fibrous thickening around the vessels, the general bulk of muscle was greater and there was no sign of hemorrhage.

Technic—Massage should be given several hours after meals and the skin should always be clean. The patient should be in the recumbent position, which permits the greatest possible relaxation. The operator should personify cleanliness and neat-

ness. Supple and warm hands are preferable to great strength. Hard massage that calls forth a protective contraction of the muscles should be regarded as an error in technique.

The various movements of massage are divided into four groups: *Effleurage*, *pétrissage*, *tapotement*, and vibration. All these movements should be toward the heart when the mechanical action is desired. When the reflex action is called for it does not matter which direction one chooses.

Effleurage, or stroking, should be done with the whole palm of the hand and should always begin any massage treatment. It can be given superficially or deep. For the latter a complete relaxation of the part treated is necessary.

Pétrissage, or kneading, is the type of massage known to athletes and commonly given in Turkish baths. It is usually deep. Relaxation of the muscles is essential, and if this is complete, no force or vigor is required for fullest possible benefit. The tissue is kneaded between the thumb and index-finger, or between the flat surfaces of both hands.

Tapotement is any hacking, clapping, or beating performed with the ulnar border or palmar surface of the hand, or with the half-closed fist.

Vibration, which may be fine or coarse, is used manually over certain delicate areas, especially the face. Electrically driven vibrators are now widely used in place of the manual effort.

Prescription for Massage—*Gentle massage*, usually *effleurage*, should be ordered in all cases of paralysis. If deep or vigorous massage is given, dilatation and stagnation of the vascular system may result. Recent injuries and other sensitive conditions call for gentle massage.

Medium Massage—Most chronic conditions except paralysis should receive medium massage. *Pétrissage* is the predominant hand movement.

Deep Massage—This type has a very limited use. In contractures, adhesions, and myositis it may be advisable. Its use is confined largely to training rooms and Turkish baths.

Contraindications for Massage—It is obvious that massage

should not be used in acute septic or acute inflammatory processes, neoplasms, injuries to the skin or superficial structures, or in myositis ossificans

Proper Use of Massage in Therapeutics—The maximum effect of massage is obtained when its use is preceded by some agent producing hyperemia, such as heliotherapy or hydrotherapy. Moist heat is probably preferable, but is not always so convenient to use as radiant light.

Mechanotherapy and Exercises—If we consider pain as a defensive reaction of the human body it does not seem logical to submit an acutely inflamed joint to the excruciating pain which accompanies exercises. Instead, complete rest, often in plaster of Paris, is indicated. On the other hand as soon as the acute stage has passed, something must be done to preserve the function of the joint and of the muscles that act on it.

Formerly, elaborate machines were constructed for this purpose, either on the principle of vibratory movements or passive exercises, but such machines are very expensive, and the physical helplessness of a patient with chronic arthritis is so reflected in his mental make-up that the idea of being fixed in any machine produces an undesirable psychologic effect. Manual or active exercises are, therefore, preferable.

The type of exercises will, of course, vary with the individual patient. To prevent atrophy of the hands, fingers, or wrist a soft-rubber ball may be squeezed, playing the piano is likewise beneficial. For the elbow and shoulder, elastic or spring exercisers are useful. The lower extremities may be exercised on a stationary bicycle.

The Swedish movements, or muscle training, are widely used and afford excellent results if there is co-operation between patient and operator. It must be borne in mind that wherever exercises with a purpose are possible (occupational therapy), they are preferable. These systematic exercises should be carefully graded, and followed by massage and rest. If, following the exercises, a patient experiences so much pain that it keeps him awake at night, the indications for less vigorous exercises are clear.

Sanitarium Treatment—Rheumatism in some form is probably responsible for the existence of most sanatoria, built usually near a spring which contains small amounts of mineral salts. The directors of these sanatoria early recognized the benefit of physical means other than water, and by close supervision of diet and nursing they obtained results where the family physician had failed. For the future sanitarium treatment of arthritis we shall probably have to look to heliotherapy in a moderately high dry altitude and even climate, and the use of such physical means as massage, diathermy, static electricity, hydrotherapy and exercises, in addition to other medical and surgical procedures. It is probable that the remarkable results obtained in tuberculosis of the joints by heliotherapy, traction, and suspension can be duplicated in the case of chronic arthritis.

Treatment of Arthritis with Physical Therapy—Having discussed the various forms of physical therapy, we may now proceed to a consideration of its application to the various forms of this disease.

Proliferative Arthritis—At the beginning of this article we divided arthritis into proliferative and degenerative types. Proliferative arthritis is inflammatory arthritis and is nearly always of infectious origin. In the early stages the inflammation is acute. In the later stages the inflammatory tissue takes on a chronic proliferative character and there is considerable increase in fibrous tissue.

When the inflammation is periarticular physical therapy is of the greatest value. In the acute stages complete rest of the joint, or joints, is indicated, and heat in its various forms gives great relief to the patient. Radiant light or a heater in the shape of a bridge is the most convenient way of applying external heat.

Diathermy is an excellent anodyne if much pain is present. Diathermy is applied with two equally sized electrodes on either side of the joint or by the cuff method where one electrode in the form of a cuff encircles the limb above the joint, and another below it. If several joints are involved, as in the lower extremities, one large electrode is placed over the sacrum and connected with the machine, while another is placed under the sole

of each foot. These sole plates are connected with each other and then with the machine by a cable. The milliamperage is stepped up slowly to 1000 to 2000 milliamperes, according to the size of the plates, the penetration, and the patients' tolerance. The treatment is continued for thirty to forty-five minutes and is then terminated slowly. The external heat can be repeated as often as desired. One diathermy treatment each day is usually sufficient, but two may be given if necessary.

In the more chronic cases the aim of treatment is to hasten the absorption of the newly formed tissue, to preserve the function of the joint, and to prevent deformity. Heat, both external and internal, is indicated as in the acute cases, but other agents should now be added. Massage is of great importance because of its muscle preserving effect and general tonic action. It should be given both generally and locally. The massage should be only moderately vigorous, consisting mainly of effleurage and pétrissage. The patient's impression after massage should be that of stimulation and not of muscle soreness. The static spark and static wave are of great value in periarticular arthritis. The physiologic action of these applications is a compression of all the compressible tissues, and they are therefore of great value in periarticular swelling. With the proper technic the use of the static machine may take the place of massage in chronic arthritis. As soon as treatment is started the masseur should introduce passive movements, very gently at first, but increasing in vigor with the patient's tolerance. In all cases of periarticular arthritis it is important to encourage active voluntary exercises, squeezing a soft-rubber ball in the hands, or generalized exercises performed to music.

A typical prescription for periarticular arthritis would be as shown on page 302 (Fig. 68).

As the fibrous tissue contracts a moderate degree of deformity, usually in the form of a contracture, may take place. This is often accentuated by atrophy of the surrounding muscles, which upsets the balance of power between the various muscle groups. This tendency to contracture must be strongly combated by massage, passive and active movements, and if necessary by

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splints and various forms of traction. Sometimes it is desirable to have the patient wear the splint only at night during sleep, and to remove it during the day.

When the articular surface itself is involved the condition is always more serious, as there is great danger of ankylosis. In

I

THE HOSPITAL FOR RUPTURED AND CRIPPLED

PHYSIOTHERAPY DEPARTMENT			
DATE	NAME	NUMBER	
DIAGNOSIS <u>Periarticular arthritis</u>			
ELECTROTHERAPY			
GALVANISM			
CONTINUOUS CURRENTS			
POLE TO		TO POLE TO	
AUTOCURRENTS			
PARADISE			
OF STEW-COIL			
SINUSOIDAL			
FLOWERS BAND			
HIGH FREQUENCY			
(2) SHAW-WERT	25'	Joint involved	500 X 2 CM
WARM			
AUTOCURRENTS FROM			
OTHER LOCAL			
STATIC			
(3) W. V.		Joint involved	
WARM			
SHOWN ON SKIN			
HYDROTHERAPY			
CARNEY			
WATER BATH			
WATERBATH			
HELIO-THERMOTHERAPY			
(1) BARK OF LAMP	15'	Joint involved	50 inches
BARKING			
DEEP THERAPY LAMP			
ULTRA VIOLET			
AND OTHERS			
ON THE SKIN			
MASSAGE			
(3) <u>Involved</u>		Extremely involved	
ON THE SKIN			
MECHANOTHERAPY AND GYMNASTICS			
(4) <u>Involved</u>		Joint involved	
MOVING			
MOVING			
GENERAL EXERCISES			
MOVING			

No. 68

Fig. 68

the acute stage absolute rest is indicated. To obtain this it is usually necessary to immobilize the joint in a plaster cast or traction is applied to prevent contact between the inflamed articular surfaces. Heat in all forms is very acceptable to the patient.

In chronic articular arthritis heat is also of great value, and in patients with good hearts the radiant light or heater may be replaced by the electric cabinet bath

Diathermy relieves the pain and by inducing a deep hyperemia prepares the joint for active or passive exercises

The static spark and wave are of great value in this form of arthritis. It is safest to start both the spark and wave with a very short distance between the discharging terminals, and to increase the distance as the patient becomes used to the treatment.

The massage should be general, even if only one or two joints are involved. It should be of medium force, consisting mainly of effleurage and pétrissage, and should leave a stimulating after-effect. The massage should follow the exercises, which should be adapted to the patient's age and general condition.

In long-standing cases general radiation with ultraviolet rays is the best tonic. We use the fractional application. The distance should be about 40 inches, and the first exposure only about two minutes, variations depending upon the lamp used. The first day only the feet should be exposed. The legs are added the next day, and the third day, the feet, legs, and knees. On the fourth day, the feet, legs, knees, and thighs, and so on until the whole body is exposed.

Arthritis deformans comes under the head of proliferative arthritis, but it is really a combination of articular and peri-articular inflammation. In many cases it does not pass through a frankly acute stage, but progresses insidiously from one joint to another until it has involved the whole body. Arthritis deformans is probably one of the most difficult of human ailments to treat. The entire co-operation of the patient is absolutely essential to success.

Because of the number of joints involved the electric cabinet bath should be used whenever the patient's heart permits it. It should be given at a temperature of 140° to 160° F until a profuse perspiration results. It should be followed by a fan douche or alcohol rub. The electric cabinet bath should be given only two or three times a week, as they are weakening

splints and various forms of traction. Sometimes it is desirable to have the patient wear the splint only at night during sleep, and to remove it during the day.

When the articular surface itself is involved the condition is always more serious, as there is great danger of ankylosis. In

I

THE HOSPITAL FOR RUPTURED AND CRIPPLED

PHYSIOTHERAPY DEPARTMENT			
DATE	NAME	NUMBER	
DIAGNOSIS: <u>Periarticular arthritis</u>			
ELECTROTHERAPY			
GALVANISM	TIME	PART	STRENGTH
CONTINUOUS-IONIZATION		+POLE TO	
INTERUPTED			
PARADISE			
BAINBRIDGE			
SINUSOIDAL			
SLOW-MED-RAPID			
HIGH FREQUENCY			
(2) DIATHERMY	25'	Joint involved	900 ± mamp
ODIN			
AUTOCOMPRESSION			
GENERAL-LOCAL			
STATIC			
(3) WAVE		Joint involved	
SPARK			
BRUSH OR BRIDGE			
HYDROTHERAPY			
CAB NET			TEMPERATURE
SCOTCH DOUCHE			
WHIRLPOOL			
HELIO-THERMOTHERAPY			
(1) RADIANT LIGHT	16'	Joint involved	50 inches
BATHING			
DEEP THERAPY LAMP			
ULTRA VIOLET			
AIR COOLED			
WATER COOLED			
MASSAGE			
(5) REVOLVING DEEP		Extremity involved	
VIBRATION			
MECHANOTHERAPY AND GYMNASTICS			
(4) ACTIVE		Joint involved	
PASSIVE			
MEDICAL GYMNASTICS			
SANDWICH			

PH-8-24

M D

Fig 68

the acute stage absolute rest is indicated. To obtain this it is usually necessary to immobilize the joint in a plaster cast or traction is applied to prevent contact between the inflamed articular surfaces. Heat in all forms is very acceptable to the patient.

Arthritis deformans being a chronic progressive disease the tonic effect of the ultraviolet radiation is indicated. It should be applied every other day until a good tanning is obtained. Ultraviolet radiation being only a poor substitute for heliotherapy, the sun treatment should be preferred wherever possible. We look forward to the construction of a lamp giving the whole sun spectrum, *i e*, the heat, luminous, and ultraviolet rays. Such a lamp is not on the market as yet, but would be of enormous value in therapeutics.

A typical prescription card for arthritis deformans would be as shown on page 304 (Fig. 69).

Gonococcus arthritis is usually periarticular, but may involve the whole body. It manifests itself in all degrees of virulence and all degrees of severity. The milder forms are easily controlled by gonococcus vaccine and the application of physical therapy in the form of heat, diathermy, massage, and passive movements. In the severe forms immobilization and traction may be necessary.

Degenerative arthritis, or hypertrophic arthritis, is characterized by a new growth of bone in and around the articular surfaces. In addition to this the capsule is often thickened. It is best exemplified in monarticular arthritis of the hip, sometimes called morbus coxae senilis. Senile arthritis, the arthritis of menopause, and Heberden's nodes also fall in this group. Strangely enough, a considerable degree of hypertrophic arthritis can exist in a joint without causing any subjective symptoms. This is well illustrated by Heberden's nodes, which are frequently painless, and by the hipping of the lumbar vertebra so frequently observed in middle-aged persons who are entirely unaware of the condition. This fact should give encouragement to the physician who attempts to treat arthritis of this type. It is obviously impossible to remove the bony exostoses by any mode of physical therapy.

The only thing we can hope for is to relieve the pain. This is accomplished by external heat and diathermy. The diathermy electrodes are applied so that the joint is in the direct path between the plates. The diathermy should be given from

They should be followed by static spark or wave for ten minutes and general light massage

Passive and active exercises play a great part in this group. As the patient has to persist in them for a long time it is important to make them either useful or interesting. In large hos-

II

THE HOSPITAL FOR RUPTURED AND CRIPPLED

PHYSIOTHERAPY DEPARTMENT			
DATE:	NAME:	NUMBER:	
DIAGNOSIS: <u>Arthritis deformans</u>			
ELECTROTHERAPY			
GALVANISM	TIME	PART	STRENGTH
POLE TO		+POLE TO	
INTERRUPTED			
FARADISM			
BRISTOW-COIL			
SINUSOIDAL			
SLOW MED.-RAPID			
HIGH FREQUENCY			
DIATHERMY			
QUIN			
AUTOCONDENSATION			
GENERAL-LOCAL			
STATIC			
WAVE			
(3) SPARK		joint involved	
BRUSH OR BREEZE			
HYDROTHERAPY			TEMPERATURE
(1) CABINET	10-15°		140-160°
(2) SCOTCH DOUCHE	Fan douche - General		
WHIRLPOOL			
HELIO-THERMOTHERAPY			DISTANCE
RADIANT LIGHT			
BAKING			
(6) DEEP THERAPY LAMP	10°	Gen. 50°	50 cm.
ULTRA VIOLET			
(7) R COOLED	5°	Gen. 50°	60 cm.
WATER COOLED			
MASSAGE			
(4) MONT OMEG-DEEP	20°	General	
VIBRATION			
MECHANOTHERAPY AND GYMNASTICS			
(5) ACTIVE		General	
PASSIVE			
MEDICAL GYMNASTICS			
(6) BANDS		General	

UN-9-24

M D

Fig 69

pitals it is possible to take a group of similar cases together in a class. Mass movement exercises are most successful in advanced cases, and it is often wise to teach a patient while in bed to go through a routine of exercises while a march is being played on a victrola.

Arthritis deformans being a chronic progressive disease the tonic effect of the ultraviolet radiation is indicated. It should be applied every other day until a good tanning is obtained. Ultraviolet radiation being only a poor substitute for heliotherapy, the sun treatment should be preferred wherever possible. We look forward to the construction of a lamp giving the whole sun spectrum, *i. e.*, the heat, luminous, and ultraviolet rays. Such a lamp is not on the market as yet, but would be of enormous value in therapeutics.

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The only thing we can hope for is to relieve the pain. This is accomplished by external heat and diathermy. The diathermy electrodes are applied so that the joint is in the direct path between the plates. The diathermy should be given from

twenty-five to forty-five minutes and the maximum amperage that the patient can stand. In a hip-joint with plates 5 square inches it is safe to give 1200 milliamperes. Local massage is indicated after the diathermy, but if exercises are given they should be only active ones. Any effort at passive movements for such a joint is usually followed by severe and lasting pain.

The proper technic for diathermy in these cases is the application of the two plates opposite each knee-joint. The two median plates are connected with each other and the lateral ones to the machine. We have also good results from one cuff electrode below each knee connected with the machine. For the treatment of the fingers a cuff electrode is applied to each forearm lower third and the patient is instructed to press her fingers together.

Senile arthritis and menopause arthritis also belong to this group and for some reason respond to physical measures more readily than do the monarticular cases. It often happens that the middle-aged obese woman with pain and crepitation in knees and fingers is relieved of her symptoms after the first treatment. The effect of diathermy in menopause arthritis is really striking. It should be preceded by external heat ten to twenty minutes, and followed by massage.

In hypertrophic arthritis ankylosis does not occur. There is a gradual erosion of the cartilage, and finally the underlying bone may become exposed, so that bone actually comes in contact with the bone of the opposite articulation. This wearing away of the articular surfaces gives rise to the grating or crepitation so frequently noted with this type of arthritis. As the lipping becomes more marked there may be considerable limitation of motion because of these bony outgrowths, and it is doubtful if any form of physical therapy can restore function thus impaired. The most that we can do for these patients is to make them more comfortable by relieving symptoms and checking the progress of the process.

A typical prescription for hypertrophic arthritis would be as shown on page 307 (Fig. 70).

Prevention of Deformity in Arthritis—From the very onset

of the disease the possibility of later deformity should be kept in mind and every effort should be made to prevent it. If this is impossible, the joints should be kept in a position that per-

III

THE HOSPITAL FOR RUPTURED AND CRIPPLED

PHYSIOTHERAPY DEPARTMENT			
DATE:	NAME	NUMBER:	
DIAGNOSIS: <u>Menopausal arthritis, both knees</u>			
ELECTROTHERAPY			
GALVANISM	TIME	PART	STRENGTH
CONTINUOUS-ION BATH		± POLE TO	
POLE TO			
INTERMPTED			
PARADISE			
BRISTLE-COIL			
SINUSOIDAL			
FLOW MED-RAPID			
HIGH FREQUENCY			
(2) DIATHERMY	30'	Both knee joints	900 mamp.
ODDS			
AUTOCOMPRESSION			
GENERAL-LOCAL			
STATIC			
WAVE			
SPARK			
SPARK ON SKIN			
HYDROTHERAPY			TEMPERATURE
CABINET			
SCOTCH DOUCHE			
WHIRLPOOL			
HELIO-THERMOTHERAPY			DISTANCE
RADIANT LIGHT			
(1) BAKING	20'	Both knees	
DEEP THERAPY LAMP			
ULTRA VIOLET			
A R COOLED			
WATER COOLED			
MASSAGE			
(3) GENTLE-DEEP	10'	Both low extremities	
BRATION			
MECHANOTHERAPY AND GYMNASTICS			
ACTIVE			
PASSIVE			
MEDICAL GYMNASTICS			
SAUNDER			

IM-5-34

M.D.

Fig 70

mits the greatest usefulness. The means at our command may be considered under four headings:

1 *Active and passive exercises* can be performed by the patient himself, by a technician, or by machines. In the case of active exercises by the patient the opponents of the contracted muscles should be worked to the point of fatigue. The machines constructed for active exercises on the principle of

the pendulum movement produce contraction and stretching of both extensors and flexors on a joint.

Passive exercises performed by a technician are probably the most satisfactory method. From the earliest possible stage of the disease the motions in the joints should be practised once or twice a day. It is useful to combine stretching of the contracted muscles and tendons with contraction of the opponents by means of resistance exercises.

2 *Plaster*—Just as we rely on plaster to prevent deformity in poliomyelitis, so it may be used in deforming arthritis. In arthritis the plaster casing should be bivalved early to permit other treatments. Often it is best to leave the patient free for motion in daytime and apply the bivalved plaster only at night.

3 *Braces*—Well-applied braces can be substituted for plaster, and have the advantage that they are easily removed and can be constructed so as to permit stretching of the contractures and contraction of the opponents.

4 *The Horizontal and Balkan Frame*—The horizontal frame, also called the Bradford frame, may be used in spinal arthritis when the spine is rapidly going on to ankylosis. The patient lies on his back on this frame with the desired curve in the lumbar region and when the patient's spine is completely ankylosed it is in an upright position instead of in the well-known semi-circular form.

The Balkan frame with traction and suspension, as popularized by Blake for the treatment of fractures, lends itself admirably to the treatment of arthritis. By means of traction the joint surfaces are kept apart and the weights can be so balanced that very little effort is required to move the joint. In a knee-joint the overhead weight can be made heavier in order that it may exert a steady pull on the contracted flexors.

The horizontal frame and the Balkan frame can be combined. This method has another advantage in that it does not interfere with heat, massage, diathermy, etc.

Limitations of Physical Therapy—There are very few cases of chronic arthritis that will not be benefited at least to some extent by physical therapy. On the other hand, it would be a

great mistake to look upon this valuable method of treatment as a cure-all

In the preceding paragraphs we have tried to indicate the physiologic basis for the various forms of physical therapy. In the more acute forms it relieves the pain, reduces the congestion and hastens the absorption of the inflammatory exudate. In the chronic forms physical therapy again acts as an anodyne, increases the blood-supply to the part, accelerates the local and general metabolism, loosens up adhesions, and stimulates the muscles supplying the part.

In degenerative arthritis physical therapy is of great value in softening the tissue, in preventing atrophy and loss of function, and by various mechanical devices in preventing or relieving contact.

The limitations of physical therapy are several. In the first place, no permanent cure can be expected of physical agents until the cause of the disease has been discovered and removed. In rare instances nature heals the original focus and the disease undergoes spontaneous recovery, but the physician who waits for nature to heal the foci of infection will usually be disappointed.

2 In advanced ankylosis of a joint, either fibrous or bony, the benefits obtained from physical therapy are limited. In the fibrous form, however, it is surprising how much can be accomplished toward restoration of function even in those cases where motion is extremely limited.

3 In cases where marked deformity has already occurred it will usually be found impossible to restore the joint to its normal form. A good illustration of this is the Heberden node. Once the finger is bent it is almost impossible to straighten it out again.

4 Physical therapy will rarely remove bony exostoses. As a matter of fact, early exostoses can undoubtedly be absorbed by diathermy, properly applied, but the old bony outgrowths so commonly seen in menopause arthritis and monarticular arthritis of the hip-joint cannot be removed by any of the methods at our disposal. It should be borne in mind, however,

that a joint is not necessarily painful because the articular surface shows hypertrophic changes. For this reason every patient with a degenerative form of arthritis is entitled to physical therapy. Most of the patients will be considerably helped by the treatment.

5 The success of physical therapy is dependent in large measure on the skill and experience of the operator. It is very unfortunate that in most instances the physician knows nothing about physical therapy and the operator knows nothing of medicine. The physician cannot expect to get the best results with this form of therapy until he has acquainted himself with the various methods and with the technic of their application.

Physical therapy differs in no respects from other forms of therapy, it can be successfully employed only when it is skillfully employed.

Finally, it is to be remembered that in those cases that are not permanently cured by physical therapy often experience great temporary relief from the various physiotherapeutic agents. It is doubtful if a physician is ever justified in telling an arthritic patient that nothing can be done for his condition. Even the advanced cases are entitled to whatever comfort they may be able to obtain from physical treatment.

A CLINICAL LECTURE BY DR HARLOW BROOKS

CITY HOSPITAL, NEW YORK CITY

LIVER DISEASE CAUSED BY HEART DEFECTS

A FEW days ago a case of acute glomerular nephritis was admitted to my service at the City Hospital. The man was alcoholic, and had been indulging in the Volstead types of alcoholics liberally and recently. The urine was loaded with albumin, with red blood-cells and many casts, hyaline, granular, blood and epithelial. The blood retention was moderately high, but the excretion of phenolphthalein was only slightly defective, the blood-pressure was 220/110. He was moderately edematous, there was a small amount of ascites, very little dyspnea, a little edema of the lungs at the bases, but his chief complaint was of a sense of distention in the epigastrium, flatulence, and of a dull but severe ache in the region of the liver. The liver was greatly enlarged, four fingers below the costal border, very tender and sensitive. He was pale, his hemoglobin was but 55 per cent, he was not particularly dyspneic, and not cyanosed. He complained almost exclusively of the pain and discomfort in the region of the enlarged liver and of his digestive discomforts. Examination of the heart showed no arrhythmias, no murmurs, the borders could not be satisfactorily determined. There was no more dyspnea than seemingly accounted for by the edema and the hypertension.

Quite naturally, I think, we drew the conclusion that in addition to the nephritis we were dealing with an alcoholic cirrhosis of the liver, which is very commonly associated in nephritis of this type. It is also well recognized that hepatic inflammation is frequently associated in acute glomerular nephritis.

We were unable to satisfactorily outline the borders of the heart so we requested an x-ray plate of the chest. The plates

showed us an unsuspected and a marked dilatation of the heart, notably of the right ventricle. We added to our Karella diet, and to our other routine designed to correct the renal defect, large doses of digitalis. Within a week the liver border was at the costal margin, the flatulence, the epigastric pain, and the slight jaundice previously present had all disappeared. Our patient was now very comfortable, anxious to get out of bed, and to return to his home. The x-ray study showed the heart had decreased nearly 5 cm. in its borders. We discontinued the digitalis, and allowed our patient to remain on substantially the same treatment otherwise. Within a few days the old epigastric disturbance had reappeared, the liver border was palpable two fingers below the costal margin, slight jaundice was reappearing. Digitalis soon restored him to relative comfort.

Just one other instance of the liver disturbances which may arise as a result of unsuspected heart defect. Of course, had the patient been up and about the ward, we should have noted circulatory distress, cyanosis in all probability, poor quality heart sounds, and the like, but he was not up, he was in bed, and the cardiac phase of the problem had escaped us, the dominant complaints were those of liver disease.

A little, old woman was admitted to the female ward showing ascites, general edema, in the urine low specific gravity, a trace of albumin, few occasional hyaline casts. There was no particular retention of the nitrogenous bodies in the blood. The liver was very much enlarged, tender, and she was slightly jaundiced. This increased as the days went by. The heart sounds were of poor quality. The area was not enlarged, but the organ was small, even for her small body. The arteries in general were markedly sclerotic and calcified, there were no retinal hemorrhages, but the retinal arteries were tortuous, narrow, and calcified. The blood-pressure was low, 100/80, the heart sounds weak. She failed to improve under digitalis, given until nausea was produced. The signs of liver distress progressed, more pain, more tenderness, more jaundice. She developed a pulmonary edema and promptly died.

At the autopsy we found an enormous soft, congested liver,

fatty and bile stained, the kidneys showed very little aside from the arteriosclerosis and fibrosis incident to her age. The heart was small, evidently originally long and narrow, but now acutely distended, globular, with all its chambers distended with blood. The amount of disease in the coronaries was marked, the muscle was soft, almost without tone, and the color was a deep brown. Typical brown atrophy of the heart with a secondary congestion of the liver. Small wonder that our digitalis had failed to improve our muscle action, there was little normal muscle on which to act, small wonder that calomel, magnesia, low salt and low fluid intake had failed to reduce the enlarged liver, for it represented a simple congestion with secondary fatty changes dependent almost, if not entirely, on long-standing defective action of the heart. It is very easy indeed to fail to make a diagnosis of brown atrophy of the heart and even had we made it, how might we have benefited our patient now for a lesion originated years before and in so far as I know, entirely without any help except that which rest can give, and there comes a time when rest even fails.

One more case and I shall announce my text. A large fat man weighing over 200 pounds was admitted to the service, complaining chiefly of pain in his abdomen of jaundice, nausea, exhaustion, of dyspnea, dizziness, of flatulence, and of constipation. The veins of the abdomen and those of the lower thorax were distended and their anastomosis was very abnormally apparent. He was suffering from hemorrhoids. The liver was very easily palpable even through the more or less edematous abdominal wall. Some ascites was present. The heart sounds were poor, there was marked irregularity on very slight movement, very marked dyspnea and cyanosis. The fluoroscope and x-ray plate showed a broad heart and an almost symmetric shadow showing little disproportion between the right and left sides.

He failed to improve under digitalis, under Karel diet, nor were we able to relieve his symptoms by any means whatever, even with morphin. He died with general exhaustion, with extreme edema, cyanosis, and great abdominal distention. At

the autopsy we found a large, fatty, flabby heart, thin muscle walls, moderately diseased coronaries, and a normal aorta of rather small caliber. The brain was edematous, so were all the other organs. The liver was enormous, contrary to expectation there was but little increase in fibrous tissue but it was tremendously congested and the liver lobules were atrophied, bile stained, and showed extensive fatty degeneration.

The patient had been a man of inactive habits, not a large drinker, but a generous feeder, he had not developed a cirrhotic liver, but a congested and fatty one as a result of his defective fatty heart, his faulty excretion, and his overfeeding.

Extensive and serious disease of the liver develops as a result of defective heart action not only in chronic diseases, but in many acute ones also. It is quite frequently seen in cases of convalescent scarlet fever or pneumonia, after toxic myocarditis or endocarditis in tonsillitis, and it is particularly frequent in convalescent cases of influenza. Oftentimes the heart defect is obscure, occult, perhaps because the patient is in bed, and therefore no extraordinary stress existing, he fails to show the cyanosis, dyspnea, and other usual evidences of cardiac defect. Perhaps the heart defects may be obscured by noisy breath sounds, by pericardial effusions, edematous thoracic walls, perhaps the elemental defect is a narrowed coronary, even one without the symptoms of an angina, there are, indeed, many instances in which occult cardiac disease may remain unsuspected even to those most skilled in circulatory disease and most keenly alert to its signs and symptoms. Yet it is in many of these very cases that the cardiac defect may manifest itself chiefly or perhaps even solely by liver changes.

We are all of us very familiar with the liver changes which develop in outstanding cardiac disease. Some of us forget, however, that we may have, as we frequently do, outstanding liver signs and symptoms due to an obscure cardiac defect. My purpose today is to point out this fact to you and to discuss very briefly the chief points in pathology, in symptomatology, and I shall also have a little to say concerning prognosis and treatment.

Of course, in all the cases the basic fault lies in a congestion of the liver which has developed as a result of defective action of the heart, and it is obvious for simple anatomic reasons that those changes which most quickly and most profoundly affect the liver are defects of the right heart. We appreciate the difficulty which is frequently experienced in the recognition of disease of the right heart. The defect may be a valvular one and it has long been noted that tricuspid disease, notably incompetence, is promptly manifested by an enlargement of the liver, frequently by actual pulsation of that organ.

Defective clearance of the right chambers of the heart produces immediately a damming back of the return flow from the inferior cava. The large rigid walled hepatic veins open directly into the inferior cava, hence it is readily appreciated why every elevation in pressure or congestion in the inferior cava is immediately followed by distention of the hepatic veins. These terminate, or really originate, in the central venule of each lobule from which spread out as do the spokes of a wheel the intralobular capillaries of each liver lobule. Hence it is that an elevation of pressure or a congestion in the cava is quickly reacted to by changes in every part of the liver tissue.

What subsequently happens as a result of this distention of the intralobular capillaries depends on many diverse factors. Among them are the duration of the congestion, the age of the patient, which determines to some extent the amount of cell regeneration and resistance of the body, the general resistance of the patient, and largely on the degree of reaction which ensues in the delicate connective tissue which surrounds each lobule and stretches inward in a very sensitive and delicate network.

Inevitably the pressure on the cells of the distended blood capillaries is followed by an atrophy of the liver cells. The degree and finality of this is, of course, decided by the severity of the process and the duration, while age and many other factors determine the type of reaction. Irreparable damage may be easily inflicted on the liver cells, death and necrosis may follow, and in young persons one often sees attempts at reproduction on the part of the damaged cells.

Degenerative and atrophic changes of all sorts ensue. Brown atrophy is one of the most frequent end-results. Sometimes fatty degeneration takes place and the liver may absolutely resemble the typical "nutmeg" liver of a fatty degeneration. We have very little knowledge of the changes which must take place in the intricate chemistry and physiology of the liver, certainly profound alterations in the metabolism must occur, and doubtless this may account for at least some of the bizarre and curious symptoms which appear in these cases of hepatic disease secondary to cardiac defects.

Having spoken of the changes in the liver cells, how they become atrophied and degenerated, let us consider what happens to the bile capillaries. Great and abnormal pressure is also exerted on them, proper drainage is prevented, and, no doubt, proper bile secretion (or is it excretion?), anyway the patient becomes jaundiced just as in some forms of cirrhosis or in new growth of the liver.

As a result of the pressure on the faultily constructed intra-lobular capillaries hemorrhages take place in the liver substance. The cells break down and blood detritus appears in the extravasated areas, this is beautifully shown in sections stained to demonstrate blood pigment. Some of this detritus remains where it appears, some is taken up by leukocytes and fibroblasts, and some is apparently picked up by the liver cells. In either circumstance it acts as a foreign body, and therefore as an irritant. Cell infiltration occurs, fibroblasts appear, and in the end a proliferation of the normally exceedingly delicate connective-tissue stroma of the lobule takes place. More atrophy of liver cells, more pressure on bile-ducts, more jaundice, and more defective liver physiology.

If the interlobular connective tissue is thus excited to growth, subsequently to cicatrization, an interlobular cirrhosis occurs. So it happens that almost any form or type of liver cirrhosis may be closely simulated by the changes which may appear in the liver primarily as the result of a defective heart.

With this exceedingly brief and sketchy outline of the pathology taking place in the liver as a result of a primary heart

defect, it is apparent to all of you that it is exceedingly difficult to differentiate diagnostically between liver changes caused by primary circulatory discrepancies and those other conditions which lead to the so-called essential diseases of the liver. Hypertrophic cirrhosis, atrophic cirrhosis, interlobular cirrhosis, fatty liver, brown atrophy of the liver and even syphilis and neoplasm of the liver may be, and frequently are, so closely simulated that only a phenomenal egotist can be certain of his diagnostic differentiation. As to the extreme rapidity with which these changes may take place, I need only call your attention to the extremely rapid increase in size of the liver in acute dilatation of the right heart, or to those very aggressive types of liver enlargement which are seen in defects and destructive lesions of the tricuspid valve. From the size of the organ one can determine absolutely nothing as to the probable duration of the process. The liver may increase in bulk to double its volume overnight if the cardiac defect is severe enough, on the other hand, the liver changes may be extremely gradual and they may occupy years in their evolution.

The liver may then be large or small in size, its surface may be smooth or nodular, so very much depends on the individual idiosyncrasy and reaction of the patient, on his age as well as on the duration of the process. So much depends also on the subsidiary factors concerned in any case. Is the patient a glutton? Does he select his foods indiscreetly? Does he use the concentrated forms of alcohol, especially the dangerous and horrible forms now usually obtainable? The point which I wish to particularly impress upon you is that almost any form of gross liver disease may be so closely simulated that recognition from the liver lesion alone is improbable, to say the least. Not only may these various forms of liver disease be simulated, but their actual microscopic structure may be almost duplicated by the changes of primary circulatory defect.

We have shown that the liver pathology in this status may be very closely confused with essential liver disease. How about the symptomatology? Obviously, about the same must be said. I pass over those symptoms which are probably due to altera-

tions in the intricate physiochemical processes of this greatest chemical laboratory of the human body, we really know very little of them, and when it comes to attempting an understanding of symptomatology from physiologic variations, as a result of disease, we are indeed in very deep water. Probably the intoxication symptoms which appear, the headache, the albuminuria, and other evidences of renal irritation, are but gross effects of this. The lassitude, somnolence, the melancholy which even by the ancients was connected with liver disturbances are also but evidences of these delicate and obscure processes. Suffice it to say that all the symptoms which appear in liver disease of other and essential types appear also in the forms of liver disease which occur in association with circulatory disease.

Naturally we get the same enlargement of the abdominal veins, the same abnormal and picturesque anastomoses of the superficial epigastrics and the mammary branches, varicose veins of all sorts and locations appear, hemorrhoids develop, and so on.

Among the more important symptoms, chiefly so because they are among the earlier, is a sense of tenderness and pain in the region of the liver. A sense of weight is almost always complained of early. Gastro-intestinal disturbances of all sorts follow, distaste for food, flatulence, constipation, perhaps alternating with diarrhea. The abdomen becomes enlarged, tender, the colon distended with gas, jaundice appears, and is apparently in both skin and in the conjunctiva. The eyes become suffused, the skin is dry and scaly, the patient is dyspneic, easily exhausted, he has headaches, and is depressed and melancholy.

Now as to the types of cardiac defect which are most likely to produce serious liver disturbances of the character which we have been delineating. Any sort of heart or circulatory disease which causes slow emptying of the chambers of the right heart may cause them, whether these changes be primary in the right heart, as may be the case from thrombosis or sclerosis of the right coronary artery, or where the right heart is giving way only because of the inability of the left heart to carry on its

functions in a normal way, as in mitral disease, for example, when compensation is broken

As a rule, it is very easy to recognize this fact in these obvious cases, and it is only the highly specialized specialist who limits his inquiries to only this or that region, who fails to recognize these obvious cases of liver disease dependent on heart defects. It is not these cases, however, that puzzle us or that go to the dead house without the clinical recognition of cause and defect, and therefore without proper care and treatment.

There is, however, a very large group of cardiac disorders which cause defects of this character in the liver which are not thus easy of recognition. This is particularly true in cases of occult cardiac disease and there are many cases of this kind, only the physician who faithfully and humbly follows his cases to the dead house knows how many.

In instances of obesity, where the heart sounds are obscured by abnormal deposits of fat. In bed cases where, because no stress is imposed on the circulation considerable cardiac defects may exist, without those symptoms so patently circulatory in the patient who is up and about his work. Myocarditis of all sorts may be present with but few signs or symptoms definitely indicative of its existence. This is particularly true in the case of brown atrophy of the heart or in fibrosis of the myocardium especially when either condition appears in old age, or in the course of chronic disease. Occult myocarditis or cardiac degeneration is common in or after many of the infections, notably after typhoid fever, in tuberculosis after pneumonia, diphtheria, tonsillitis, and the like. It is so common, and often so very difficult to detect after influenza, even in supposedly mild cases that this relation demands special mention.

Adhesive pericarditis of greater or lesser degree is so commonly a cause of hepatic change without definite cardiac symptoms that this requires also special attention. Libman has also called particular attention to this association with adhesive pericarditis and he has emphasized also its frequent association with sino-auricular block.

One must not fail to remember the very great frequency of

a myocarditis of very obscure type, as after scarlet fever, tonsillitis, and other forms of streptococcus infection. The occurrence of hepatic signs and symptoms are often the very first obvious evidence of this defect. Several times I have found hepatitis in an entirely unsuspected myocarditis, especially after these streptococcus infections. Libman, with others, has often pointed out the frequency with which hepatic disease appears in bacterial endocarditis.

So we might go on almost indefinitely with many obscure and difficult-to-detect types of heart disease which show liver signs and symptoms before cardiac defects are apparent, but perhaps what I have said will suffice if I conclude this phase of our discussion with the statement that in every case of liver disease, of whatever apparent cause, the heart should be carefully searched before its possible causal relationship is discarded. This requires in a good many instances the very unscientific but very effectual "therapeutic test."

Always remember that a heart murmur may mean little, or nothing, or much. It is the lesion back of the heart murmur that counts. The absence of a heart murmur is no sign of cardiac integrity, it simply means that a heart murmur is not present, that is all. A perfectly normal electrocardiographic record no more clears the heart of suspicion than an absent skin rash at any one time negatives a scarlet fever. Only a most thorough and understanding study of the heart, particularly in relation to its response to physiologic and even pathologic demands, can clear the heart of suspected occult disease. Even the autopsy is not finally conclusive and pathologic histology may fail—one can never be sure. One of the most helpful methods of cardiac examination in cases suspected of cardiac defect in these cases of liver disease has been the use of the fluoroscope. This I have found ordinarily more satisfactory than mere x-ray studies for the reason that with but very little dilatation of the left ventricle or auricle as portrayed on the x-ray plate, back of the fluoroscopic screen an abnormally active degree of pulsation of that chamber may appear very strikingly. In several puzzling instances I found this method of inestimable value.

Just a few words as to prognosis in these cases of liver disease caused by cardiac defects. As always with prognosis this is a very difficult and complicated problem. Much depends on the curability of the primary lesion in the heart or, if this be impossible, on the amelioration. Also one must always bear in mind that though it may be possible to cure the cardiac lesion, as by rest after many of the acute infections, one must consider then if the changes already established in the liver tissues may not in themselves become now fixed and irremediable. Secondary conditions in large number must be also considered. Is the patient willing to strictly follow out your diet? Will he give up his indiscreet eating? Will he stop his alcohol or his favorite sauces? We know these secondary factors to be of the greatest possible importance in these damaged livers.

It is always quite safe to say that prognosis should be held in abeyance until the therapeutic test has been made and until we are able to say definitely that the cardiac lesion is or is not a curable one, or if the changes already present in the liver may not under favorable conditions subside.

As to treatment. It is, of course, obvious that the primary essential is to attack the primary defect—the faulty heart. In the majority of cases this can be most essentially encompassed by *rest*. Rest here must comprise not merely an exclusion of all factors of physical stress. Emotional stress is almost as difficult for the heart to tolerate as physical stress or strain, hence our rest must be both physical and emotional. Rest need not necessarily imply bed, some of these patients do far better when up at least part of the time, rest must be always individually determined. It must be rest for that particular patient, not some standardized text-book bed treatment or the like designed for the group, here we must definitely individualize.

By far the larger number of these cases show the most striking good results in so far as the heart and liver are concerned when they are put upon some form of the digitalis group of drugs. In many of these cases the entire liver pathology apparently subsides under nothing but digitalis, and in suitable instances the occasional exhibition of digitalis has sufficed to

keep these patients thereafter in very good condition. As a rule, I am not partial to the use of digitalis, especially after the acute infections which have induced a myocarditis, but in these cases of hepatic involvement I am insistent upon its early employment, even though rest may eventually accomplish even more for the heart itself. This is because of the reason that the longer the liver lesion is permitted to remain, the more difficult it becomes to finally eradicate it. Oftentimes it becomes fixed as a progressive liver change, though the primary cardiac defect may have long since been eradicated. In these cases, as in most instances of long-standing cardiac decompensation, I find, as a rule, that the digitalis is best administered in courses with periods of intermission between. I have been unable to satisfactorily calibrate my dosage of digitalis by any set rule of dosage per body weight, but I have followed, particularly in these cases, the rule long since laid down by Cushny, my old teacher of pharmacology, to "give digitalis until digitalis effect is produced." That was the modern digitalis treatment advocated back in 1895, and I still find it entirely satisfactory.

One must never forget the liver in these cases. Where treatment is solely directed toward the heart, very unsatisfactory hepatic results may occur, though in a very considerable number of instances, especially the early cases, the correction of the cardiac defect may entirely suffice.

Our first effort must be also directed to the elimination of all those conditions which may accelerate the liver process. Food must be limited strictly to the needs of the body, determined somewhat by the amount of energy which the patient is required to expend. We should particularly eliminate all hepatic irritants, such as alcoholics, the spices, as pepper and aromatics, among which perhaps we should class asparagus, rhubarb, etc. The diet should not be too highly nitrogenous. If ascites or edema is present, we must restrict water and fluid foods, such as milk. Under such circumstances we must also cut down, or out, salt, and if the blood-pressure be too high, these elements must be eliminated anyway.

Another very important factor in the treatment, at least of

most of these cases, is the discreet use of purgatives. The average case, especially if combined with ascites or edema, profits well by the routine administration of the saline cathartics, and my favorite is magnesium sulphate given in some suitable form each morning. Some cases do better on sodium phosphate, magnesium citrate, and the like. Nearly all cases profit from occasional doses of calomel or blue mass. A favorite method of mine, taken from a very old custom in the profession, is the giving of the Niemeyer, Fothergill's, or St. Bartholomew's pill, three times daily, one day out of seven, or more often as may seem wise. To those of you who may not happen to be old enough to know of the pill I will say that it is composed of digitalis, squills, and calomel, of each 1 grain.

A rigid Karez diet is very useful in many cases, especially those in which ascites or edema is pronounced. Special cases will demand special treatment. In most cases showing ascites I resort early to abdominal tap, and I repeat it at as frequent intervals as the distention or discomfort indicates. I think that frequent tapping is in some cases actually curable.

Some cases show marked benefit from the diuretics, caffeine, soda benzoate, diuretin, theosin, and of late we have been using arsenural with good results. In two cases, however, I believe that this last drug, administered intravenously, had a very dangerous result. I do not think that it should be employed in cases which are not known to be insensitive to arsenic, especially in those cases which are known to have extensive degeneration of the liver parenchyma.

Massage, active and passive movements, may be well applied in some instances, but throughout it must be constantly borne in mind that the essential basis of the condition is a cardiac defect and the heart must be constantly watched. No overload of any kind is to be permitted, and drugs are to be used symptomatically whenever necessary.

Remember that the most important step in diagnosis is accomplished when the physician merely remembers that many liver lesions and symptoms may be produced by cardiac defects.

CLINIC OF DR NELLIS B FOSTER .

NEW YORK HOSPITAL

THE CLINICAL PICTURE OF CHOLECYSTITIS INDUCED BY BACTERIAL ENDOCARDITIS¹

It is well known that pneumonia may cause abdominal pain and that this pain along with localized tenderness and spasticity of the abdominal muscles before signs of pneumonia are detectable occasionally leads to errors in diagnosis. This mistake is particularly apt to occur in the pneumonia of childhood.

Abdominal pain due to cardiac disease is not so well recognized, although there are plenty of cases cited in the literature. Since it is germane to the general subject under discussion, the case which you saw at autopsy about two weeks ago is worthy of review. You recall that this patient was a woman about fifty years old who was brought to the hospital on account of pain in the epigastrium radiating to the right shoulder and back, nausea, and vomiting. There was rigidity of the abdominal muscles in the upper right quadrant. The heart gave no obvious evidence of disease but the Wassermann reaction was four plus.

The diagnosis of cholecystitis had seemed probable, but at operation the gall-bladder was found to be normal and there was no other cause for the symptoms detected. The patient rallied well after the operation and had less than the usual discomfort, but on the next day suddenly died. You recall that at autopsy we found a typical leptic aortitis and that the orifice of the right coronary artery was almost closed. It admitted only the finest probe.

This case was in no sense peculiar or rare and the condition

¹ Saturday Seminar of the Clinical Clerks.

presents ordinarily no special difficulty in diagnosis. But when the pain is referred to the abdomen rather than to the left shoulder and arm the question of some acute abdominal disease always arises and often there appears urgent need for surgical intervention. Paul White, of the Massachusetts General Hospital, has specially emphasized this clinical syndrome and its diagnostic pitfalls.

There is another type of heart disease which in some respects may resemble coronary stoppage, since abdominal pain may be the prominent symptom, and in consequence of this there is apt to be confusion with other diseases which are commonly treated surgically.

There have been 2 cases in the hospital during the last few months that illustrate the clinical picture which I now bring to your attention.

As I read the synopses of these histories I wish you would endeavor to decide what diagnosis you would have arrived at on the evidence.

For the sake of conciseness I omit some irrelevant data and give you the facts so far as they were determined.

The first case was that of a woman forty-five years old who came to the hospital on account of "stomach trouble." She had had indigestion for about a year with eructations after meals and pains radiating to the right shoulder, but never to the right hypochondrium. This pain had been persistent, never very severe, nor of the nature of colic, but severe enough to disturb her rest and to destroy her appetite. She had lost 20 pounds in weight, which she assigned to the inability to eat a normal amount. There had been neither nausea, vomiting, nor diarrhea. She got no relief from various forms of medication. In the past she had had rheumatic fever twenty years ago and some milder attacks since. Several years ago she was admitted to Roosevelt Hospital during one of these attacks of rheumatic fever, and it was then determined that there was a positive Wassermann reaction and she was treated for syphilis for an indefinite period. This patient was not jaundiced, although her skin had a yellowish tint like that of pernicious anemia. There were

no abnormalities in the skin. Definite signs of aortic insufficiency were present. The liver was enlarged, the edge being felt just above the umbilicus. The surface of the liver was smooth and the spleen was palpable. The admission diagnosis was chronic valvular heart disease, aortic insufficiency, and probably luetic aortitis. It was suspected that the abdominal symptoms might be due either to disease of the gall-bladder or to early carcinoma of the stomach. The absence of hydrochloric acid along with occult blood in the stomach contents and a moderate degree of secondary anemia seemed to point toward the diagnosis of neoplasm. The Wassermann reaction was again found strongly positive. During the period of observation the patient's temperature ranged between 101° and 103° F. An investigation of the intestinal tract by means of the fluoroscope did not disclose anything significant. Abdominal pain continued and on account of the persistence of fever it was determined to do an exploratory operation. The gall-bladder and all of the accessible organs were found normal. A blood-culture, taken just before this operation, was reported four days later as containing a non-hemolytic streptococcus. Subsequently other blood-cultures confirmed the first and the course of the disease afterward was typical of bacterial endocarditis.

It is interesting that, although hunted for, there was no evidence of emboli or petechia until a month after the patient was admitted to the hospital.

The second case was a woman fifty-two years old who had had recurring attacks of pain in the stomach for seven or eight weeks before admission. The pain was localized just about the umbilicus, was continuous and burning in character, and not influenced by food. On account of the pain appetite had decreased. There was no vomiting or nausea and the patient had noted no abnormality in either stools or urine. She had lost about 18 pounds in weight. She gave no history of similar attacks in the past and she had always had sound digestion. The past history was unimportant. There was no history of any significant acute infection or symptoms of cardiac, vascular, or renal disease.

On examination the patient was somewhat flushed, the skin was a good color, and there was no jaundice. The heart rate was 96 and there was a gallop rhythm with a presystolic murmur audible in the third and fourth spaces inside the apex. The blood-pressure was low, about 96/60. The abdomen was full, tympanic, and tender in the upper part where the impression was gained of deep resistance. In the right lower region one got the impression of an indefinite mass. The liver was palpable 4 cm below the costal margin, the edge sharp and well defined, and the surface smooth. The spleen was not palpable. There was fever of 102° F and leukocytosis of 12,600. The urine contained large amounts of albumin, casts, and white blood-cells, but no blood. The Wassermann was positive. Again the symptoms suggested gall-bladder disease with the possibility of early carcinoma of the stomach. Fluoroscopic examination was indefinitely suggestive of carcinoma of the stomach since a filling defect was observed, though it was recognized that this might be due to spasm. There was no free hydrochloric acid in the stomach contents. On the day following admission it was decided that there was a mass palpable in the gall-bladder region and an exploratory operation was decided upon. At operation the mass was found to be an enlarged Riedel's lobe, the gall-bladder was normal and all of the other organs accessible apparently were normal. Blood-cultures were made on account of the persistence of the fever, *Streptococcus viridans* grew in the culture and this was confirmed by subsequent cultures from the blood.

The patient died in the hospital and necropsy showed that the tricuspid orifice was almost occluded by a large vegetation. There were also vegetations in the wall of the right auricle and a few small ones on the mitral valve. There was no occlusion of the coronaries and but slight aortitis.

The criteria for the diagnosis of bacterial endocarditis are fever, signs of embolism, and evidence of endocarditis. The diagnosis is proved by the culture of the blood. In fully developed cases the diagnosis is not difficult provided the evidence is looked for. The disease, however, may exist for months before the patient regards himself as sufficiently sick to consult his

physician, hence there is usually a history of gradual decline in health and then some episode such as a severe attack of vertigo, or acute arthritis, or chill and fever. In cases of chronic valvular heart disease, with fever, we always suspect the possibility of this type of secondary infection, it is seldom engrafted on sound hearts. For bacterial endocarditis is not a rare disease, during the last ten years we have had 1 case to every 3 cases of typhoid. It is a disease every internist and surgeon must look for.

The abdominal pain which was the striking symptom in the 2 cases under discussion is not uncommon at some time during the course of the disease. Usually it is produced by infarctions, most often in the spleen, over which a friction-rub may sometimes be heard, sometimes in the kidneys causing blood in the urine. Rarely we have found mycotic aneurysms at autopsy. But sometimes there is no satisfactory explanation for the pain discovered. It is possible that in these cases the condition of the heart is the cause, though we are apt to think of pain referred to the abdomen as rather restricted to aortic disease, aortitis, aortic insufficiency and sometimes angina pectoris. But occasionally in cases of myocardial disease with no detectable valve involvement nor coronary occlusion there may be quite severe abdominal distress.

Besides the summary by Osler, who was much interested in this disease you should read Libman's papers and the Review in Medicine by Blumer.

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CLINIC OF DR ARTHUR L HOLLAND

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GASTRO-INTESTINAL NEUROSES

It is quite safe to state that a considerable percentage of patients who consult the general practitioner because of indigestion do not suffer from organic changes in the gastro-intestinal tube, that their symptoms result from functional irregularities of tonus, motility, or secretion, or are the expressions of sensory disturbances that have neither a functional nor an organic basis outside of the nervous system. It is difficult, however, to divide arbitrarily all cases of indigestion into two groups: organic and nervous. Disturbances or excesses of the emotions may be important etiologic factors in organic digestive disease. On the other hand, the depressing mental effect of an organic digestive disease may result in functional derangement, the symptoms of which will overshadow those due to the lesion.

I shall not attempt at this time to discuss cases in which the gastro-intestinal canal gives expression in symptoms to reflexes from organic lesions in other organs. These might be called secondary neuroses, but being dependent on organic changes they can hardly be classified with nervous indigestion, even though the exciting lesion is in no way a part of the gastro-intestinal canal.

Before proceeding, I shall ask you to recall and to keep in mind some peculiarities of the digestive system.

By means of its autonomic nerve control the gastro-intestinal tube is ordinarily able to maintain a regular peristaltic sequence. Through reflexes and hormones the peristalsis of the various sections of the tube are made to synchronize with each other and with the functions of the accessory organs of digestion.

The maintaining of tonus or posture in all sections of the tube is another of the functions of the autonomic vegetative nervous system. These nerves have been demonstrated as possessing a duality of action, one concerned in motion (kinetic impulses), the other in posture (static impulses). The secretions also depend on impulses from the same nervous system, by direct stimulation, reflexes, or hormones. The gastric secretion, however, is regulated somewhat indirectly by the gastric motility and the motility of the tube beyond the pylorus. With a motility retarded at or just beyond the pylorus, the quantity of gastric secretion and its acid content increases. With accelerated gastric motility the gastric secretion and its acid content is reduced. We know but little about the intestinal secretions in this connection.

The gastro-intestinal tube is not sensitive to ordinary forms of irritation such as pinching, burning, or cutting. This insensibility to irritation is difficult to comprehend when it is remembered how much pain can be experienced in some forms of indigestion. This, however, is explained by the fact that the tube is sensitive to differences in its tension. Being poorly differentiated and not provided with fibers for the sensing of ordinary pain impulses, these nerves in some way not well understood register a protest, which is relayed to the brain by the spinal nerves, when any part of the tube suffers injury that results in increased tension, or, in the absence of injury, through inflammatory or mechanical agents, increased tension alone will be sufficient excuse for such a painful sensation. The degree of increased tension is probably the measure of the acuteness or severity of such a pain.

By means of the posturing mechanism, gastric and intestinal tonus is normally maintained in a remarkably even manner. This means that the intratubular tension remains relatively fixed, and any considerable deviation from a tension or intratubular pressure that is peculiar to any section of the tube results in symptoms no matter what the cause of the imbalance of tension may be. When the cause lies in an organic lesion such as gastric or duodenal ulcer, for example, the symptoms

occur with some regularity, because the demands made upon the posturing mechanism by the motor efforts of the organ during digestion are considerable, and if embarrassed by an adjacent organic lesion the pain protest which results naturally bears a relation to some phase of the digestive cycle

Through various reflexes the motor mechanism of parts of the tube may be affected. For example, a diseased gall-bladder may reflexly cause an increase in gastric tonus or, acting through a similar reflex, such an inflamed or irritable gall-bladder may cause spasticity of the descending colon and the colicky pains of colitis thus experienced. The ileopyloric reflex may cause spasticity of the gastric antrum and pylorus. This is frequently observed in lower right quadrant disease and explains the epigastric pain that is not infrequently complained of in this condition. These reflexes, on the other hand, may operate differently by bringing about a release of tension. Lowered gastric tension is most often experienced as nausea, particularly when the release is sudden, when it is of long duration or permanent, the syndrome of atonia is presented.

With a normal structure and an efficient secretory equipment the gastro-intestinal tube, through reflexes from the emotional centers or the senses, may be thrown into confusion, desynchronized, and behave quite as badly as though irritated locally or reflexly by an organic lesion. It is this extreme sensitiveness of the digestive organs that renders the study of the gastro-intestinal neuroses important. It helps to explain supposed food idiosyncrasies. It also explains the occurrence of pain where there is no inflammatory or other lesion present. It is easier to think of such pain as the result of the increased tension of spasm than of its existing only in the imagination. Actual pain, however, is not the most frequent complaint of these nervous dyspeptics. They suffer much from sensations of pressure and distention. This is attributed by them to a prolonged stay of food in their stomachs, to fermentation, or to chemical reactions of food combinations. These are, of course, misconceptions. An imbalance of intratubular tension brought about through spasm or erratic peristalsis can be made to explain

most of the symptoms that are presented in these neuroses, excepting, of course those symptoms that are themselves secondary to the erratic motor function, such as the headaches and vertigo of spastic constipation, or the mucous production and diarrhea due to the mechanical irritation of a spastic colon

It is not entirely true that neurasthenics are "born, not made" These individuals usually start life as sensitive, highly strung, emotional beings—potential neurasthenics If such potential neurasthenics be given the proper early training and education for their stations, and fall into pleasant places, or at least are protected from excessive mental shocks or strains, they may go through life being loved and honored They may even accomplish big things As a matter of fact, they are largely of the brilliant people that we know But they do not stand long-continued struggles or worries, humiliations, or repeated shocks They may appear to weather this sort of thing for a long time, but their resistance has a limit and one can never tell in a given case just when this will be reached, or in what form the psychic breakdown will manifest itself It does not usually come dramatically as the "snapping of something," spoken of in the story books, but rather insidiously

With the motor and secretory functions of the stomach depressed or accelerated as a response to an emotional upset, digestive symptoms are usually the first to be made evident, and with the patient's attention diverted from his normal and usual interests to these symptoms a vicious circle is established that soon changes the potential neurasthenic into a true neurasthenic

Nervous indigestion probably comes to the psycho-asthenic and hysteric in much the same way It is well to remember that nervous indigestion is rarely the starting-point in these troubles It usually happens and takes root in well-prepared soil An apparently normal individual may suffer temporarily from some form of nervous indigestion because of some severe strain or shock, but it will usually be found that his resistance has been lowered by some constitutional condition

Nervous indigestion should be looked upon as in no way con-

nected with the digestive apparatus except that the symptoms are made evident through these sensitive organs. Among the gastro-intestinal neuroses and very important, are the irregularities of appetite. I shall follow Déjerine's classification¹

Primary mental anorexia "consists in the progressive loss of the mental representation of appetite" (Déjerine). It may have its beginning in emotional causes, usually of a depressive nature, grief, shocks, domestic or love troubles. Regular fasting as a religious observation or fasting as a stunt has been known to precipitate it. The deprivations of poverty have caused anorexia that has been carried over into more propitious times. These patients can take but little food, they lose strength and easily fall victims to tuberculosis, anemia, and other constitutional diseases. Young women are largely the sufferers from this form of anorexia, but not exclusively.

Case I—Miss T. J., thirty years of age, American saleslady. Had always been in fairly good health, except for chorea as a child and some slight irregularity of the menstrual function. Three and one-half years ago her mother, to whom she was much attached and dependent upon, became ill with a cancer. It was necessary for Miss J. to continue with her work and at night to nurse and attend the patient. Notwithstanding the strain, she seemed to maintain her strength and health until the death of the mother, two years later. She then found it impossible to eat solid foods. She could swallow only small quantities of liquids. This has continued to the present time, even though she has been somewhat relieved of financial embarrassment through relatives and has had constant medical attention. She is a sensible young woman, has given no evidence of morbid or unstable mentality. Her only symptoms are inability to eat, a slight but progressive loss in weight and strength, and a moderate degree of constipation. The physical examination is essentially negative. Fluoroscopically the esophagus, stomach, and intestines have been found normal except for slight loss in

¹ Déjerine, *Psychoneuroses and Psychotherapy*, Trans. by Jelliffe, Lippincott & Co., 1913.

gastric tonus and a moderate spasticity of the descending colon. Gastric analysis shows a total acid of 40, free HCl 24.

Secondary mental anorexia is similar, but it is usually dependent upon some physical condition in which a reduction of diet has been thought necessary, or the patient through fear of a repetition of some unpleasant experience has limited his diet. Even though the conditions apparently warranting the low or restricted diet have been rectified, the patient finds that he has lost interest in food and cannot be induced to partake of a normal quantity of food. This state of things may obtain in cases where an effort has been made to overcome obesity, or following some temporary gastro-intestinal upset. It may be met with in both sexes and at all ages.

Neurasthenic individuals are particularly prone to this form of anorexia.

Case II—Mr. M. B., twenty-eight years of age, American draughtsman. Had always been quite well until four years ago, when he suffered a small gastric hemorrhage. Melena persisted for about a week. Previous to this he had never had any definite gastro-intestinal symptoms. Gastric ulcer was diagnosed and he was placed on a regular ulcer treatment. He recovered in due time, but persisted with a liquid or a very soft diet for two years. A complete examination at this time proved negative and he was advised to extend his diet to include all things except those obviously contraindicated, such as tough meats, uncooked vegetables, and uncooked fruits. This he found impossible to do. He believed in his medical adviser and wished to comply with his orders, but he had lost interest in food of all kinds and could take only those things that he could swallow with little effort. He is 25 pounds below his normal weight. He finds his light duties as draughtsman not easy because of lack of strength. He has been constipated since the onset. The physical examination is essentially negative except for the evident loss in weight, pallor of the skin, and general lack of muscular tonus. A gastro-intestinal x-ray series was negative. Gastric analysis shows acid values slightly below normal.

Case III—Mrs P. S., forty-nine years of age, American, widow Was told six years ago that she was suffering from arteriosclerosis and that unless she reduced her diet very much the disease would progress She at once complied and since then has subsisted on an incredibly small amount of food Two months ago she consulted a heart specialist, who told her that the arteries did not show any changes that are not usual for one of her age and that her loss in weight and strength, of which she complains, are due entirely to her low diet She has not been able to increase the quantity of food

Elective secondary mental anorexia is somewhat different It comes about in much the same way but has less to do with quantity It is a qualitative restriction After a course of diet from which certain foods have been eliminated, the patient cannot return to the eliminated foods even though he is assured that the conditions originally apparently warranting their elimination have been corrected He will eat sufficient of the foods that were not proscribed and his weight and strength do not suffer, but his distaste for the foods that he has long ago stopped eating cannot be overcome

Case IV—Mr L. H., thirty-eight years of age, American, shopkeeper Ten years ago he suffered headaches and other indefinite symptoms that he was told were due to autotoxemia He visited a sanitarium where meat is anathema nuts and vegetables being used to supply the necessary proteins He became a vegetarian He now cannot be induced to eat meat of any kind, not only because he considers it harmful to him but also because it nauseates him even to think of it In justice to the sanitarium and its system it should be reported that he has been free from his headaches and the other symptoms he had complained of, but he is a problem to his family whose other members may partake of meats only when he is not about the house

The diagnosis of mental anorexia is made on the history The loss of appetite in these cases can nearly always be traced to some interference with the diet, either voluntary or imposed

by physical conditions or imagined disease. Where this tampering with the diet has occurred, and the psychic idea of appetite cannot be re-established, although the exciting conditions are no longer present, the diagnosis is plain. It may, however, be difficult to differentiate mental anorexia from hysteria in which food is taken only surreptitiously. In these latter cases the weight and strength are rarely affected.

Excesses of appetite may be brought about in a similar manner to mental anorexia, for instance, through the stressing of the importance of hyperalimentation to overcome some physical shortcoming or the misconception that food must be taken at regular times or fainting will result and that mental or physical work will not be possible without it. In these cases the call of the tissues for nourishment is normal, the false appetite being purely mental.

It may be stated broadly that in primary and secondary mental anorexia the gastric secretions, if altered at all, are low in acid, but this is not an invariable rule. The acid values in the excesses of appetite are usually either normal or raised. But here one must exercise care. A hyperchlorhydria in itself will cause increase in appetite, and so it is difficult always to say just how much of the excess appetite is purely mental. As the causes of hyperchlorhydria are many, and often hidden, it becomes necessary to diagnose these neuroses by exclusion, although the histories in many of the cases are strongly suggestive.

Our responsibilities in these matters are heavy. Since ancient times physicians have felt called upon to regulate the diet of their patients and to formulate rules of "hygienic eating" for the community at large. It is not an exaggeration to state that a large part of this activity has proved not only worthless, but actually harmful, as much of it has been founded on false theories and ignorance. Unfortunately, a spirit of commercialism has often prompted individual medical men to exploit this or that faddish diet. The layman has always been and still is confused regarding these matters and his medical attendant is not always any better informed, although posing as an authority and guide. Ethically considered, this would all be regret-

table, but more far reaching in unfortunate results has been the constant stressing of unnecessary dietetic restrictions. Neurasthenia and kindred mental invalidisms too often have a "diet" as an etiologic factor, nor is such invalidism always the ultimate result. Death directly through starvation has resulted, or indirectly through the occurrence of constitutional disease made possible by a constitution weakened by insufficient nourishment.

In treating any disease, particularly the gastro-intestinal diseases, one is not justified in prescribing a diet which is not clearly indicated. The forbidding of one or another combination of foods, this or that article of diet, should be based on something more definite than somebody's theory of food chemistry in its relation to the digestive processes. In a general way we can accomplish a great deal by suggesting a proper balancing of foods, more of this element and less of that, or suggestions as to the character of food may be necessary, whether it be soft or rough, etc. But so far as possible, patients should be allowed freedom in choosing the articles of diet of which they are to partake.

In reviewing the literature of only a few years ago, one is struck by the great variety of gastro-intestinal neuroses mentioned and discussed. In the light of modern teaching, these all resolve into so many symptoms and it becomes us to treat them as such without any serious attempt at classification.

These symptoms differ but little from those caused directly or reflexly by organic diseases. The chief difference is in the irregularity of presentation, both as to their relation to meal times and to the character of the food. Unless definite phobias have been established, it seems to make but little difference what is eaten—at times heavy food is apparently well tolerated, while some simple article of diet may appear to cause distress. This tolerance and reaction may change from time to time.

The emotional mood that happens to dominate the patient at the time of eating a meal will usually determine his reaction to it. Happy and interested, his stomach will accept and tolerate a meal suitable for a woodchopper. But depressed or in the

throes of ennui, he experiences symptoms, no matter how innocent or simple the repast may be

Belching need not detain us long This is a nervous habit and is seen commonly in the neuroses But being somewhat dependent on excess gastric motor activity, it may be excited reflexly by inflammatory lesions such as gall-bladder disease or ovarian irritation Where this acquired trick is exhibited when the patient is nervously upset or only while being observed, the chances of its being due to organic trouble are less than when it occurs regularly at some time in the gastric digestive cycle

Nausea that bears some relation to meals may be dependent on any one of a number of organic changes, but as a reaction to some mental upset it is more likely to be of nervous origin The same is true of vomiting, although a great deal of nervous vomiting occurs immediately after meals or even while the patient is eating He may leave the table, vomit, and return to complete his meal This type of vomiting is seldom accompanied by retching, sweating, or blanching of the skin The food is gulped up and ejected with very little effort

The vomitus often gives reliable information Mucus means some gastritis Food from meals that should have been discharged from the stomach long before suggests stasis Blood tells its own story A hypersecretion type of vomitus, *i. e.*, a large quantity of clear, sour, watery material, with or without bile, is usually due to spasticity at or beyond the pylorus It therefore suggests a reflex irritation such as chronic appendicitis, adhesions, ovarian disease, etc A large amount of vomitus having a butyric odor tells of gastric stasis If food, eaten many hours previously, is seen in such a return, one may be sure of it Prolonged pyloric spasm or atony may, of course, be responsible, although it is rare to find food remains from previous meals in the gastric returns in atony

Epigastric burning, heart-burn, and pyrosis are the commonest forms of distress complained of by nervous dyspeptics It rarely bears any relation to the acidity of the gastric juice It does not occur with the regularity of ulcer pain, although soda

and food may relieve it temporarily, but not regularly. They may appear to increase the trouble.

A sense of epigastric weight or fulness is also a common symptom. The patients usually mentally visualize the cause of this fulness in a mass of undigested food weighing down their stomachs, "like a lump of lead," they complain.

There is no such thing as gastralgia. Such severe pain as was formerly thought to be due to what was called "gastralgia" is undoubtedly caused by reflex spasm of the cardia or pylorus, or are reflex pains from some organic disease or lesion of the central nervous system. Gastric hyperesthesia is usually the expression of hyperperistalsis, increased gastric tension, or some such motor disturbance. The stomach is not supplied with sensory nerves.

Nervous patients complain much of rumbling and other evidence of peristaltic unrest. It may occur only as a sensation or become evident through the sound of the gas being shifted from the stomach to the intestines or from one section of intestine to another. It is rarely experienced as visible peristalsis. Unless the patient is very thin, visible peristalsis is a sign of some obstruction.

Abdominal distention is also a frequent complaint of these dyspeptics, but this is more apt to be a sensory disturbance, not to be confirmed as actual distention by the examiner.

Flatulence and the discharge of gas from the rectum at defecation and at other times are symptoms much dwelt upon by neurasthenics. They are much concerned if they are thus annoyed, but, on the other hand, if they are unable to negotiate such a passage of gas they complain.

A discussion of the neuroses in relation to constipation, colitis, and such conditions involving the intestinal canal will not be indulged in at this time. In passing, the writer would state that he believes colitis is of all the neuroses the most common, and that it is, in a majority of cases, a pure neurosis, that its etiology rarely includes any organic element. Beginning as a constipation brought about through laziness, lack of exercise, drug taking, or some temporary trouble such as hemorrhoids.

these patients are persuaded that their difficulty is of an organic character. A colon thus embarrassed becomes irritable, spasm results, drugs and enemas increase the irritability. Mucus is poured out as a protection. The spasm increases, and by its pressure along with the pressure of irritating fecal masses, ulceration results. The fallacy of treating these cases by long courses of high irrigations, vaccines, and the implantation of cultures should be obvious.

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ALKALOSIS

FOR a number of years acidosis has been a well-recognized condition. You are all familiar with the acidosis of diabetes, nephritis, that following anesthesia, and that accompanying the diarrhea of infancy. The various types are well differentiated, the diagnosis can be rendered positive by laboratory tests, and the treatment is fairly well standardized. The reaction of the blood is, however, so nicely balanced that it is not uncommon for patients to whom alkalies are being administered for the relief of acidosis to develop the opposite condition—alkalosis. The significance of this has only recently been recognized, and I shall therefore devote the hour to a consideration of this condition, and to the presentation of several groups of cases.

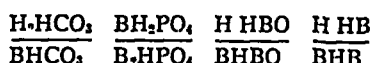
The *symptoms of alkalosis* simulate those of tetany: twitching of the muscles, particularly those of the face (if unrelieved, convulsions develop), nausea, vomiting, and hiccups, dryness and redness of the skin, edema of the extremities, vertigo and weakness, drowsiness which progresses to coma, and anuria, which may cause a fatal outcome. The condition is often recognized as a result of laboratory findings. The progress of the disease can be followed both clinically and by frequent examination of the CO_2 combining power of the blood.

The chemistry of the condition is rather technical, and Dr John A. Kilham, who supervises all our chemical analyses, has very kindly consented to present this phase.

THE CHEMISTRY OF ALKALOSIS

Chemically, alkalosis may be defined as an increase above normal of the bicarbonate content of the blood plasma. The

reaction of the blood during health is maintained within normal limits by buffer systems, of which the following are well known



The concentration of phosphorus in the plasma is much less than in the red cells, and this element plays a relatively unimportant part in regulating the reaction of the plasma. The oxyhemoglobin and hemoglobin also are not concerned directly with the maintenance of the normal acid-base balance of the plasma. The reaction of the blood plasma is primarily dependent upon the ratio $\frac{\text{H}\cdot\text{HCO}_3}{\text{BHCO}_3}$. All base not combined with fixed acids in the blood plasma is in the form of BHCO_3 . With the tension of CO_2 which exists in the plasma any alkali introduced into the body is converted into bicarbonate. CO_2 is found in the plasma in two forms: (1) Dissolved as H_2CO_3 , and (2) combined with base as bicarbonate. The CO_2 contained in H_2CO_3 comprises 3 volumes per cent, and that in bicarbonate form, about 60 volumes per cent. Hence,

$$\frac{\text{H}\cdot\text{HCO}_3}{\text{BHCO}_3}, \quad \frac{3}{80} \quad \frac{1}{20}$$

The reaction of the blood plasma is dependent upon this ratio. So long as the ratio remains within the limits of $\frac{1}{20}$, the P_h of the plasma will be within 7.35 to 7.43.

An alkalosis may be produced by increasing the value of the denominator of the fraction, or by decreasing that of the numerator. If, in either case, the ratio changes from $\frac{1}{20}$, the P_h will be increased above 7.43. Such a condition is an uncompensated alkalosis. However, if the body maintains the ratio $\frac{1}{20}$, the P_h will remain within normal limits, and this condition is known as compensated alkalosis.

The CO_2 combining power of the blood plasma (or the CO_2 content) is a measure of its bicarbonate content. However, to form an adequate concept of the alkalosis, it is essential to know both the CO_2 combining power and the P_h of the plasma.

PRESENTATION OF CASES

I am fortunate in having here today 2 cases of alkalosis to show you. The first patient has had a perfect recovery, but the second is in a very serious condition.

Case I—Henoch's Type of Idiopathic Purpura, Presenting Symptoms of Alkalosis¹—This school boy (S S), aged thirteen years, entered the hospital April 22, 1925 because of vomiting which had persisted for sixteen days. Aside from the usual diseases of childhood, and a brief attack of influenza eight years before, his general health had been good. Sixteen days before admission he began to vomit forcibly, and continued to do so at least twice a day. Occasionally, however, he retained a meal. There were no associated symptoms and no fever. He did, however, become constipated and, beginning twelve days prior to admission, enemas were given daily. The day before admission he raised thick mucus streaked with bright red blood. There was no history of bleeders in the family.

On physical examination it was found that the scleræ had a slight icteric tint, and that there was slight tenderness and voluntary muscle spasm in the right upper quadrant. Purpuric petechiæ were also noted.

The laboratory findings are given in Table 1 (p. 346).

An x-ray of the gall-bladder and one of the genito-urinary tract were negative. The temperature, pulse, and respirations were normal.

Repeated urinalyses showed the specific gravity to average 1012, there was considerable protein, no sugar, a few hyaline and granular casts, many red blood-cells, and a few white blood-cells. The reaction varied from alkaline to acid, but was more often alkaline.

May 1st the symptoms of purpura increased, the possibility of gall-bladder complications was suggested, and a surgical consultant called. It was decided, however, that the condition did not require surgical intervention, and that the vomiting was probably a compensatory effort at elimination. A provisional diagnosis of septicemia was made, and blood transfusion advised.

¹ Pediatric Service, Dr. Marshall Pease.

The clinical course during the patient's stay in the hospital, the chemical findings, and the treatment instituted are summarized in Table 1. Under the treatment indicated, infusions of 10 per cent glucose in normal saline solution, dilute HCl administered by rectum (inasmuch as the patient was vomiting), and Murphy drip of glucose and normal saline, the patient's condition steadily improved, the vomiting ceased, and the alkalosis was relieved.

In this case the purpura led to the vomiting, and the vomiting to the alkalosis.¹

TABLE 1

CASE I CLINICAL COURSE, TREATMENT, AND CHEMICAL FINDINGS

	CHEMICAL ANALYSIS OF BLOOD					VOMITING ²
	Creatinin	Urea	Chlorids	CO ₂	P _a	
April 23d	10.6	75.1	0.438	63.4		++
" 25th		40.8	0.413	74.0	7.6	++++
" 26th	2 oz dilute HCl per rectum 500 cc 10 per cent glucose in normal saline given					
" 27th	2 infusions of 500 cc, 10 per cent glucose and normal saline given day before, dilute HCl m 10 t i d, 2 oz dilute HCl by rectum. Purpura developed					
" 28th	9.3	49.6	0.388	78.6	7.54	+++
	Patient greatly improved, still vomiting slightly dilute HCl m 10 t i d, 2 oz per rectum					Receiving
" 29th	Patient on same treatment					
May 1st	Surgical consultation					
" 2d	Tonic and clonic convulsions of face and arms, vomiting, nystagmus, opisthotonus, patient critically ill					Transfusion given

¹ Since the lecture, the patient died with nephritic symptoms. It was shown at necropsy that hemorrhages had occurred throughout the kidney and intestinal tract.

² On a scale of 0 to ++++

Case II —Alkalosis Secondary to Persistent Vomiting Caused by a Ventral Hernia—This patient (G R) came to the hospital April 5, 1925 because of unexplained severe hiccups and moderate intermittent vomiting of one week's duration. His condition was serious. Physical examination revealed, in addition to the symptoms already mentioned, a marked edema. The vomiting was found to be due to a ventral hernia, and the alkalosis was attributed to the vomiting. The patient's CO_2 combining power was 92.2, indicating a very severe degree of alkalosis. The blood chlorids were 0.269—about one-half normal, the blood urea was 44 and the P_a 7.55. All the symptoms indicated a marked alkalosis.

The day following admission an infusion of 1000 c.c. of 1/10 normal saline solution was given, also 20 minims of dilute HCl by mouth. May 7th the dose was increased to 140 minims, 1000 c.c. of 1/10 normal saline solution was given by hypodermoclysis. May 8th 140 minims of HCl were administered as before, and 200 c.c. of 1/10 normal saline solution and 10 per cent glucose solution were given by Murphy drip. Under this treatment the chlorids gradually increased to 0.513, and the CO_2 combining power decreased. May 9th the CO_2 combining power had dropped to 66.6, the blood chemistry approached normal, and the symptoms had practically subsided. It was nevertheless considered advisable to continue the treatment for several days, and 140 minims of dilute HCl were given as before until the 14th. One more infusion of saline and glucose was given on the 10th.

The diet was restricted to fluids from the 5th to the 9th, but following that a high carbohydrate diet with an ample amount of salt was allowed.

April 20th the patient left the hospital in good condition. He was so grateful for being cured that he was very willing to come to the lecture this morning. You may judge for yourselves how well he looks physically.

I have, in addition to these 2 patients, a series of 50 cases of alkalosis,¹ 25 of which I have selected to discuss briefly.

¹ Several of these cases have been on the services of Drs. Erdmann, Shattuck, Heald, McCarthy, and Mosenthal and are published with their permission.

today For convenience I shall divide them into six groups as follows (1) Alkalosis following Sippy treatment, (2) alkalosis following alkali therapy, (3) alkalosis after persistent vomiting, (4) alkalosis associated with intestinal obstruction, (5) alkalosis with fever, and (6) ketosis associated with conditions of alkalosis

GROUP I ALKALOSIS FOLLOWING SIPPY TREATMENT

In the Sippy treatment for ulcer the patient is given about 200 grains of bicarbonate of soda daily in 20-grain doses This is sufficient to produce alkalosis in many patients, especially those with a lessened ability to eliminate alkalis Four such cases are represented in Table 2 These occurred during the course of Sippy treatment for gastric and duodenal ulcer The high P_h in Case II and the lowered chlorid content of the blood in Case III should be noted

TABLE 2
ALKALOSIS FOLLOWING SIPPY TREATMENT

BLOOD ANALYSES						Remarks	
Case	Age	CO ₂ C P vols per cent	P _h	Chlorids as NaCl per cent	Sugar per cent		Urea N, mg per 100 c c
1 J B	45	75*	7.40				Gastric ulcer After forty-eight hours of treatment
		66*	7.49				After twenty-six days of treatment.
		68	7.50				After twenty nine days of treatment
2 M S	61	70	7.53				Gastric ulcer After nine days of treatment
		70	7.53				After ten days of treatment
		56	7.52				After three days of no treatment.
3 J C	39	72		0.388	0.080	23.7	HB = 16.8 Duodenal ulcer after seven days of alkaline therapy
		62		0.400		25.0	Alkaline therapy discontinued for four days six days after gastro- enterostomy
4 L K		77					Gastric ulcer After Sippy treat- ment

* CO₂ content

GROUP 2 ALKALOSIS FOLLOWING ALKALI THERAPY

The next group is one of 5 cases in which alkalosis developed following the administration of alkaline drugs in conditions other than ulcer (Table 3). It was in such cases that our attention was first called to the overtreatment of patients with alkali.

TABLE 3
ALKALOSIS FOLLOWING ALKALI THERAPY

BLOOD ANALYSES.						Remarks.	
Case.	Age.	CO-C.P vol. per cent.	Ph.	Chloride as NaCl. per cent.	Sugar per cent.		Urea No mg per 100 c.c.
1 T.A.	4	33 125		0.563		100	Bronchopneumonia. Anuria Twenty-four hours after 30 gm NaHCO ₃ intravenously
2 D.C.	43	52 72 83 100 100 81			0.374 0.316 0.264		Diabetes. After NaHCO ₃ for forty-eight hours. After NaHCO ₃ for ninety-six hours. After NaHCO ₃ for one hundred and eighteen hours. Alkali discontinued for forty-eight hours. Anuria, death after tetanic spasms. HCl given by mouth.
3 L.C.	62	76	7.60	0.475	0.225		Diabetes. After alkali therapy ketonuria, edema, and death.
4 M.L.	12	67* 70*	7.52 7.52	0.438	0.103	18.3	Cyclic vomiting. Clinical diag- nosis of acidosis. Alkali therapy. vomiting persisting for eleven days.
5 M.B.	50	73		0.425	0.366	23.4	After alkali therapy for suspected acidosis. Ketouria. Coma and death.

* CO₂ content

The first case is that of a child suffering from bronchopneumonia, with an acidosis of 33. On the basis that 0.5 gm of bicarbonate of soda will raise the CO₂ combining power of the blood 1 per cent., in a patient weighing 42 pounds, it was estimated that 30 gm of soda bicarbonate was necessary to overcome the acidosis in this case. However, the administration of this amount threw him into deep alkalosis (125) with anuria and death ensued. The reason for this unexpected reaction was

that he had impaired renal function (100 mg of urea for each 100 c c of blood), and could not eliminate normally. This case illustrates the danger of giving large doses of alkalis to patients with renal involvement.

The second is a characteristic case of alkalosis with tetanic spasms, developing after the administration of alkalis to a patient with diabetes, supposed to be suffering from severe acidosis. You will note that he had a large increase in the CO_2 combining power, and that the sugar content dropped. He developed anuria.

The third case is one of diabetes with an acid urine, due to ketosis treated by alkalis, but the blood showed a CO_2 combining power of 76 and a P_{H} of 7.6.

The fourth case illustrates the common mistake of making a clinical diagnosis of acidosis in a case of cyclic vomiting. The chemical examination of the blood shows that the patient did not need alkali. The reason for his developing alkalosis is that by vomiting he had depleted the system of hydrochloric acid. When a patient is vomiting so intensely that the hydrochloric acid administered by mouth cannot be retained, we give it by rectum with olive oil, or with physiologic salt solution directly into the vein.

The fifth case shows the danger of diagnosing acidosis upon finding acetone bodies in the urine.

The cases in this group developed alkalosis after treatment for suspected acidosis.

In Groups 1 and 2 the alkalosis has been produced by increasing the value of the denominator by the extraneous administration of alkali. As previously stated, any alkali administered tends to increase the bicarbonate content of the plasma. The effect of alkali therapy upon the blood will depend upon two factors: the rate of absorption of the alkali, and the rate of its elimination. If the absorption is slow, the body is enabled to decrease the rate of elimination of CO_2 by the lungs, so that both the numerator and the denominator are increased, but the ratio remains 1:20, hence, there is a normal P_{H} . This is a condition of compensated alkalosis, and is characterized by a

high figure for the CO_2 combining power, but a normal P_h . If, however, the rate of absorption is rapid, as in intravenous therapy, or if the elimination of alkalies by the kidneys is subnormal, the body is unable to compensate for the increased bicarbonate content of the plasma by retaining CO_2 . Hence, the ratio will no longer remain 1 : 20, and the P_h will be increased. There results an uncompensated alkalosis, having a high CO_2 combining power and a high P_h . Case I in Table 1 was at the outset one of compensated alkalosis, but after continued therapy the alkalosis became uncompensated.

GROUP 3 ALKALOSIS ASSOCIATED WITH INTESTINAL OBSTRUCTION

It has been known for some time that alkalosis frequently develops in cases of intestinal obstruction in which the obstruction is rather high up. This is due to the loss of hydrochloric acid by vomiting, and the retention of alkalies, owing to the impairment of renal function so often occurring in this condition. The increased amount of urea in these cases is significant. It has been customary in many cases to administer alkali following gastro-enterostomy for duodenal ulcer, and also in case of shock, and sudden rise of temperature. The danger of doing this is shown by the evidences of alkalosis in the cases in Table 4. The first patient was given glucose and saline solution to counteract the untoward effects, but in spite of this developed tetany and died. In these cases it is interesting to note the drop in the chlorid content of the blood as the CO_2 combining power increases, and the reverse of this condition following saline infusions. The beneficial effect of saline infusions is well shown particularly in Cases VI and VIII.

When chlorine is lost from the body it is lost principally as hydrochloric acid. This leaves in the blood plasma available base which combines with H_2O and CO_2 to form bicarbonate. We have here an increase in the value of the numerator indirectly through the chlorids. It is interesting to note in these cases that although the chlorids of the blood are markedly diminished, the sodium is but slightly decreased. The sodium released by the

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TABLE 4
ALKALOSIS ASSOCIATED WITH INTESTINAL OBSTRUCTION

Case	Age	BLOOD ANALYSES					Remarks
		CO ₂ C P vols per cent	Pb	Chloride as NaCl per cent.	Sugar, per cent.	Urea N, mg per 100 c c	
1 A C	56	86	7 58	0 525	0 204	87 4	Duodenal ulcer with pyloric stenosis two days after gastro-enterostomy T = 101.2° F Coma
		53		0 788	0 225	96 3	Twenty four hours later, after glucose and saline infusions Spasms Died
2 W C	37	98	7 48	0 263	0 156	83 3	Hb = 16.1 Intussusception with newgrowth Tetany T = 103.8° F Persistent vomiting Died
3 M K	35	88 105	7 54	0 320	0 163	56 2	Obstruction due to new growth Persistent vomiting T = 102° F Tetany Twenty four hours after entero-enterostomy Died
4 M W	67	79		0 375	0 178	20 1	New growth of pancreas obstructing duodenum and gall bladder Vomiting
		76		0 495	0 194	15 4	After forty-eight hours glucose and saline infusion
		75		0 475			Twenty four hours after cholecyst gastrostomy and gastro-enterostomy Died five days later
5 S K	58	78		0 413	0 097	30 2	Obstructive duodenal ulcer Vomiting
		62		0 413	0 178	12 8	Seven days after gastro-enterostomy
6 M L	43	78		0 400	0 157	31 7	Hb = 17 Ca of sigmoid with obstruction Three days after cecostomy Persistent vomiting T = 102° F
		86			0 170	39 1	Hb = 15.5 Six days P O Vomiting Twitching
		91	7 52	0 375		49 5	Eight days P O Vomiting HCl by mouth
		65 5		0 488		23 7	After six days of saline infusions.
		50 4		0 550		25 8	Hb = 4.5 Vomiting ceased
		57 0		0 563		9 5	Hb = 4.5 Three days later
		53 8		0 538	0 136	9 1	Hb = 7.6 After transfusion. Improved
7 F W	35	83			0 220	89 3	Paralytic ileus after hysterectomy Coma Spasms of face No vomiting
		106		0 325		52 3	Five hours later Died
8 G R	50	92		0 269	0 123	44	Partial intestinal obstruction Vomiting and hiccups Alkaline gastric lavage retained
		78	7 55				Twenty four hours later Saline infusions
		91		0 488	0 125	23	Forty-eight hours later Saline infusions
		41		0 450		9 2	Nine days later Patient improved
		57		0 475		9 9	Six days later Patient improved

chlorine is converted into bicarbonate. Not only is chlorine lost from the body, but, in addition to this, the ability of the body to replenish its supply from that of the diet is diminished. Rarely do we see compensated alkalosis in these cases, because the condition has progressed beyond this stage before the patients enter the hospital. The use of saline therapy is well founded upon the chemical changes noted in the blood.

GROUP 4 ALKALOSIS AFTER PERSISTENT VOMITING

Five cases without definite intestinal obstruction, in which alkalosis has been produced as a result of depleting the body of hydrochloric acid by vomiting, are shown in Table 5. The mechanism of the production of the alkalosis here is similar to that described for cases of alkalosis following intestinal obstruc-

TABLE 5
ALKALOSIS AFTER PERSISTENT VOMITING

Case	Age	BLOOD ANALYSES					Remarks.
		CO-C P vols per cent	Pa	Chlorids as NaCl p-r cent	Sugar, per cent	Urea N mg per 100 c.c.	
1 E.S.	75	88		0.425	0.115	37.5	Five days after prostatectomy. Persistent vomiting. Abdominal distention.
		34				31.7	Nine days later. Improvement after saline infusions and HCl by mouth.
2 A.F.	58	79		0.425	0.100	21.8	Vomiting for three days after hysterectomy.
3 B.H.	62	41			0.410	59.6	Diabetes mellitus. Five days after prostatectomy. Abdominal distention. T = 100° F.
		80			0.478	63.6	Vomiting persistently for three days. T = 99.6°-100° F.
		85			0.357	57.2	Vomiting persistently for six days. Edema and twitching of extremities. Semicoma. Death two days later.
4 V.L.	65	63	7.52		0.116	42.4	Chronic nephritis. Vomiting frequently for fifteen days.
		58	7.40				Vomiting ceased four days previous.
5 S.G.	30	60			0.196	19.4	Acute nephritis. On admission.
		72.7				93.5	Persistent vomiting for ten days.
		80				87.2	Twitching. Persistent vomiting for thirteen days. Spasms. T = 99°-100° F. Died.

tion It is important to observe, however, that vomiting is not essential to the production of alkalosis The accumulation of the gastric secretions in the stomach without vomiting may result in similar changes in the blood

GROUP 5 ALKALOSIS ASSOCIATED WITH FEVER

In cases of fever, owing to the increased respiratory rate, the patient throws off too great an amount of CO_2 and there is a resultant lack of balance between the bicarbonate of soda and the carbonic acid in the blood, the ratio 1 : 20 being disturbed by reduction of the value of the numerator It is important to note in these cases (Table 6) that the CO_2 combining power of the blood is not necessarily increased

TABLE 6
ALKALOSIS ASSOCIATED WITH FEVER

Case	Age	BLOOD ANALYSES					Remarks
		CO ₂ C P, vols per cent	P _H	Chlorids as NaCl per cent	Sugar per cent	Urea N mg per 100 c c	
1 J A	43	84		0.575	0.234	104	Operated for acute gangrenous gall bladder Postoperative infection Saline infusions T = 101°-104° F Died
2 S A	47	79 77		0.413	0.163	72 33.7	Nephrectomy for pyonephrosis Nine days P O T = 102°-103° F for seven days Five days later T = 103°-105° F Died
3 D M	37	67	7.56				Postoperative Hepatic abscess T = 102.2° F
4 R S	10	82			0.151	20.6	Encephalitis, cardiac decompensation T = 102° F Twitching of facial muscles
5 F H	41	761	7.49				Cholecystectomy P O infection T = 102° F

¹ CO₂ content

GROUP 6 KETOSIS ASSOCIATED WITH CONDITIONS OF ALKALOSIS

Table 7 represents 8 cases, the most interesting ones from the standpoint of the general practitioner It has been thought that an acid urine precluded the possibility of alkalosis The

TABLE 7
KETOSIS ASSOCIATED WITH CONDITIONS OF ALKALOSIS

Case	BLOOD ANALYSES			Remarks
	CO ₂ -C.P., vols per cent.	P _H	Acetone bodies, mg per 100 c.c.	
1 A.C.	84	7.58	60	Carcinoma of stomach Fever
2 F.G.	83	7.54	50	Carcinoma of stomach, persistent vomiting
3 M.K.	88	7.50	66	Intestinal obstruction due to malignancy
4 N.V.	87	7.52	28	Alkali therapy after cholecystectomy
5 L.K.	77		8	Gastric ulcer Sippy treatment
6 A.F.	79		17	Persistent vomiting after hysterectomy
7 M.W.	77		10	Persistent vomiting, after cholecystectomy
8 F.W.	106		11	Paralytic ileus after hysterectomy

above 8 cases all had acid urine with a marked increase in the acetone bodies of the blood, together with a high degree of alkalosis. The total ketones of blood as acetone range from 0.2 to 1 mg per 100 c.c. In addition to the CO₂ combining power the P_H was high. If the examination of the urine alone had been relied upon, these cases of alkalosis would have been overlooked. Fat is incompletely oxidized in the body, either when the blood is too alkaline or when it is too acid. Another cause for an acid urine in the presence of alkalosis is the retention of alkalies, owing to impaired renal function. The above cases illustrate the importance of blood-chemistry examination. Alkalosis may produce the odor of acetone on the breath, also acetone bodies may appear in the urine.

A probable cause of this ketogenesis may be the starvation secondary to the repeated vomiting, or the intestinal obstruction. Moreover, the existence of this ketogenesis indicates the value of the administration of glucose in cases of alkalosis.

TREATMENT

The treatment of alkalosis consists in giving dilute hydrochloric acid by mouth. If the patient is vomiting, 1 ounce of dilute hydrochloric acid in 2 ounces of olive oil may be given by enema twice a day. Beneficial results have been obtained by giving 500 c.c. of 10 per cent. glucose, in normal saline solution, intravenously. The markedly beneficial results of this form of

treatment are shown by the alleviation of the patient's symptoms, and the chemical findings in the blood. The CO_2 combining power of the blood is lowered, and the chlorid content is increased. Normal saline may be given by hypodermoclysis, and glucose and saline are given in retaining enema. The condition of the blood will indicate the frequency with which these means should be employed.

CLINIC OF DR GEORGE G ORNSTEIN

COLUMBIA UNIVERSITY*

PULMONARY SYPHILIS

THE one factor that plays so great a part in doubting the probability of clinical syphilis of the lung is the inability of the pathologist to histologically differentiate between a gumma and a tubercle when it is found in the lung. This perhaps accounts for syphilis of the lung occurring as a rare autopsy finding. Pulmonary syphilis is more frequently thought of clinically than the following autopsy figures warrant. Fowler¹ found but twelve specimens in the museums of the London Hospitals and the Royal College of Surgeons. Two of these cases were doubtful. At the Johns Hopkins Hospital Osler² reported 12 cases out of 2800 autopsies. Of these 12, only 4 were acquired. Among 3000 autopsies at the Massachusetts General Hospital Lord³ found only 1 case of acquired syphilis. Symmers,⁴ in a study of 4800 autopsy protocols, 314 of which showed lesions of syphilis, reported 12 cases and syphilitic pleural scars in 2 more. Syphilis of the trachea and bronchi is perhaps a less rare condition. Connors⁵ in 1903 collected 128 cases. Symmers⁴ mentions 4 cases involving the trachea among 4800 autopsies. Lord³ reported 2 cases in 3000 autopsies at the Massachusetts General Hospital.

How may we explain this marked difference between clinical impressions and autopsy findings? The tendency has been for the pathologist to diagnose tuberculosis in preference to syphilis unless spirochetes could be demonstrated in the histologic sections. The opportunity for confusion between syphilis and tuberculosis can perhaps be demonstrated in no better way than

* Department of Tuberculosis, Vanderbilt Clinic

by quoting MacCallum's description of the tertiary stage of syphilis ⁶

"The most characteristic, though not the most common, manifestation of the effects of the spirochetes in this stage of the disease is the gumma, which received its name from its elastic, rubber-like consistency. Most commonly such nodules are found embedded in the tissue and surrounded on all sides by radiating fibrous tissue, which in itself is not especially peculiar. But the central portion is firm, elastic, opaque, and yellowish white, like hard cheese. This is the necrotic caseous part, analogous to that found in tubercles, but different in its elastic, firm consistency and in the slighter tendency to liquefy. In it one may sometimes discern faint outlines of pre-existent tissue now necrotic. The margin or capsule is often not specially characteristic, being made up of a rather dense tissue rich in epithelioid cells, such as are found in tubercles, and closely infiltrated with mononuclear wandering cells. Giant-cells with multiple nuclei, such as are found in tubercles, occur, but are rarer here. Baumgarten denies their existence in gummata, claiming that they are characteristic of tubercles, and that if they do occur it is because of a coincident tuberculous infection. Such nodules may be of almost any size from minute points as small as the smallest tubercle to huge, tumor-like masses easily felt through the abdominal walls as they project from the liver, where they seem to reach their greatest size. In the milary gummata there may be no caseation or coagulative necrosis and the nodule is seen as a more or less concentrically arranged group of epithelioid cells richly mingled with mononuclear wandering cells and occasionally with giant-cells. The arrangement is usually indefinite and irregular, lacking the sharpness and precision of the architecture of the milary tubercle. Spirochetes have been demonstrated by animal inoculation in larger gummata by Finger and Landsteiner, but they are not easily found in sections. How, then, is one to tell a gumma from a tubercle when it is found at the autopsy? Histologically, it seems almost impossible to make an absolute differentiation between them. A section through a gumma in the lung tissue might have exactly the appearance of one from a large caseous encapsulated tubercle."

There is no question that pulmonary syphilis is rare even as a clinical entity, but not as rare as the autopsy figures suggest.

The clinical picture of pulmonary syphilis is not characteristic. The symptoms simulate those of pulmonary tuberculosis, and, as a rule, a diagnosis of early tuberculosis is made. The cases referred to the clinic were all suspected to be tuberculosis.

I have the pleasure of presenting for your consideration a case of secondary syphilis of the trachea and bronchi simulating asthma, 1 of tertiary syphilis of the trachea and bronchi, and 1 case of tertiary syphilis of the lung.

SYPHILIS OF THE TRACHEA AND BRONCHI SIMULATING ASTHMA

Case I—M S, colored, male, age twenty-nine years. came to the clinic December 1, 1924

History—He complained of wheezing attacks that kept him awake at night. He also felt warm in the afternoon. Because

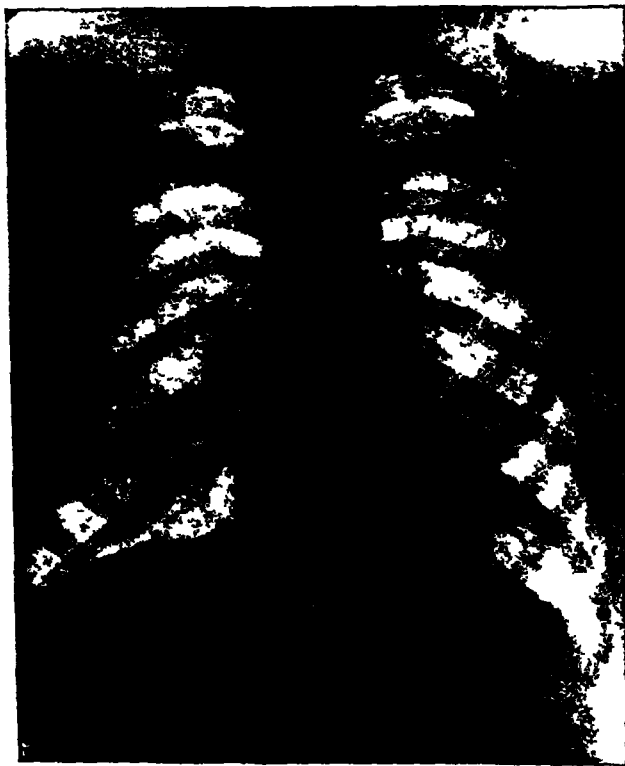


Fig 71—Case I. Except for a bilateral exaggeration of the bronchial markings, especially of those trunks leading to the diaphragm, there is no disease of the lungs.

of the wheezing and shortness of breath he has not been able to carry on his usual trade of shoe repairing. Last August he was well. At that time he had a skin eruption which was diagnosed as a syphiloderma. He was not sure when he had his chancre. He thought he might have acquired it with a gonorrhea two

years ago In August, after the appearance of the rash over his body, his Wassermann was 4+ Four weeks after the appearance of the rash he was seized with wheezing attacks which have persisted up to the present date On admission his temperature was 99° F, pulse 80, respirations 24

Physical Examination—Loud, large, sonorous sounds were heard over both lungs His heart was normal

x-Ray Examination—Except for an accenturation of the bronchial markings, the lungs were normal

The skin of his whole body showed a brownish, coppery mottling with slight scarring, probably the remains of a macular-pustular eruption On examining his mouth, over the left buccal mucosa there was a mucous plaque about the size of a twenty-five-cent piece There was congestion of the pharyngeal and palate structures The uvula was very congested and had an appearance similar to a mucous patch without the sloughing Dr John A Fordyce and Dr Fred Wise called it a "syphilitic angina" of the pharynx With such pathology in the mouth and pharynx we thought we might be able to explain the sudden appearance of these wheezing attacks to a similar condition in the trachea and bronchi We referred him to the bronchoscopic clinic of Dr M C Myerson at the Beth Israel Hospital with a diagnosis of syphilis of the mucous membrane of the trachea and the bronchi, causing asthmatic attacks

Dr Myerson's report was as follows "On the left buccal mucosa there was a mucous plaque about the size of a twenty-five-cent piece There was an acute congestion and slight edema of all the pharyngeal and palate structures The palate showed several small macular lesions His larynx showed the same type of involvement as his pharynx The bronchoscopic findings were as follows The tracheal and bronchial walls in their non-cartilaginous portions were the seat of the same type of congestion and edema as the structures just mentioned The mucosa in the cartilaginous portions of the trachea and bronchi showed slight capillary prominences here and there, with an increased redness of the mucosa On the anterior wall of the right main bronchus, about 15 mm from the bifurcation of the trachea,

an irregularly crescentic white area was seen. This could not be wiped away, nor could it be removed by suction, and was identical in appearance with the mucous plaque observed on the buccal mucous membrane. The buccal plaque was observed through the same bronchoscope for comparative study, in this way the similarity of the lesions was definitely established."

The patient was placed on intensive antiluetic therapy. There was an immediate clearing up of his asthma. The patient felt so well he decided he did not need any more therapy. He went back to his shoe repairing. Two months later he returned to the Clinic complaining of asthmatic attacks. His Wassermann was 4+. He was again placed on intravenous injections of salvarsan, with marked improvement of his asthma.

Discussion—Perhaps this may be an exaggerated form of the catarrhal manifestation that occurs in the bronchi during the appearance of the secondary rashes of syphilis. I have always been suspicious that during this stage a similar eruption occurs in the mucous membranes of the trachea and bronchi which was responsible for the catarrhal bronchitis. The bronchoscopic observation of Dr. Myerson confirmed this impression.

SYPHILIS OF THE TRACHEA AND BRONCHI SIMULATING PULMONARY TUBERCULOSIS

Case II—A. J., male, white, thirty-eight years old, came to the clinic February 12, 1925.

History—He complained of a cough for the last two years. Two years ago he had coughed up $\frac{1}{2}$ ounce of blood. On January 3, 1925 he again coughed up $\frac{1}{2}$ ounce of blood. His sputum was blood streaked for a few days following the hemoptysis. He complained of cough, expectoration, loss of weight, night-sweats, and malaise. On admission to the clinic his temperature was 98° F, pulse 68, and respirations 20. His weight was 126 pounds.

Physical Examination—There was no modification of the breath sounds or râles heard. The heart was normal.

On questioning the patient he admitted a luetic history. His primary lesion occurred fourteen years ago. An x-ray was taken

of his lungs There was an exaggeration of the bronchial markings, especially in both roots and in the upper half of the left lung Two sputa examinations were negative for tubercle bacilli His Wassermann was 3+ Tuberculosis was excluded because of his negative physical findings and a negative x-ray.



Fig 72—Case II There is a marked accentuation throughout both lungs of the bronchial markings The root regions are very exaggerated There is no evidence of any parenchymatous lesion

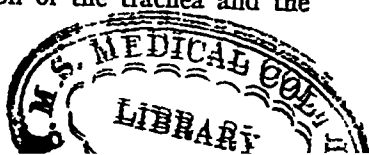
Because of a similar experience with 6 other cases,⁷ syphilis of the trachea and bronchi was thought of A tertiary syphilitic ulceration of the trachea or bronchi might easily account for the hemoptysis, the cough, and the expectoration The mild toxemia might easily be due to his syphilis The patient was

sent to the service of Dr Arrowsmith at the King's County Hospital There he was bronchoscoped by Dr M C Myerson At the King's County Hospital he was x-rayed again His Wassermann was also repeated Their reports were as follows The Roentgen examination of the chest showed nothing abnormal The Wassermann reaction was 4+

Bronchoscopic study revealed the following On the anterior wall of the right main bronchus just beyond the carina there was an irregular swelling of moderate degree This area was dark red in appearance, while the remainder of the bronchus appeared darker red than normal This area of swelling was not obstructive It measured approximately $1\frac{1}{2}$ cm in length and 5 mm in width There were two distinct white areas of ulceration, irregularly round, about 3 mm in diameter, and situated upon this area of swelling In the left main bronchus, just at the carina, there was a similar small ulcer surrounded by an area of redness, situated upon the anterior wall The diagnosis of gumma of the bronchus with beginning ulceration was rendered

Discussion—The above case and the 6 others reported in 1924⁷ gave almost a uniform history Hemoptysis accompanied with cough, expectoration, loss of weight and symptoms of a mild toxemia were the chief complaints A picture of pulmonary tuberculosis was presented to the examiner The negative physical and x-ray examinations suggested an early pulmonary tuberculosis The pathology was apparently not extensive enough to produce either physical findings or x-ray shadows There are such cases of pulmonary tuberculosis in which the physical findings and Roentgen markings appear two or three months following a history that is suggestive of the disease In the above cases the history of lues with a positive Wassermann reaction threw a different light on the diagnosis

We may summarize as follows A history of hemoptysis, cough, expectoration, etc., in the face of a normal lung condition, as demonstrated by physical and x-ray examinations, should suggest a possible syphilitic ulceration of the trachea and the bronchi



TERTIARY SYPHILIS OF THE LUNG

Case III—E D, female, colored, thirty years of age, came to the Vanderbilt Clinic, June 6, 1924

History—She had been ill for the past three months, complaining of a continuous cough with a moderate amount of ex-

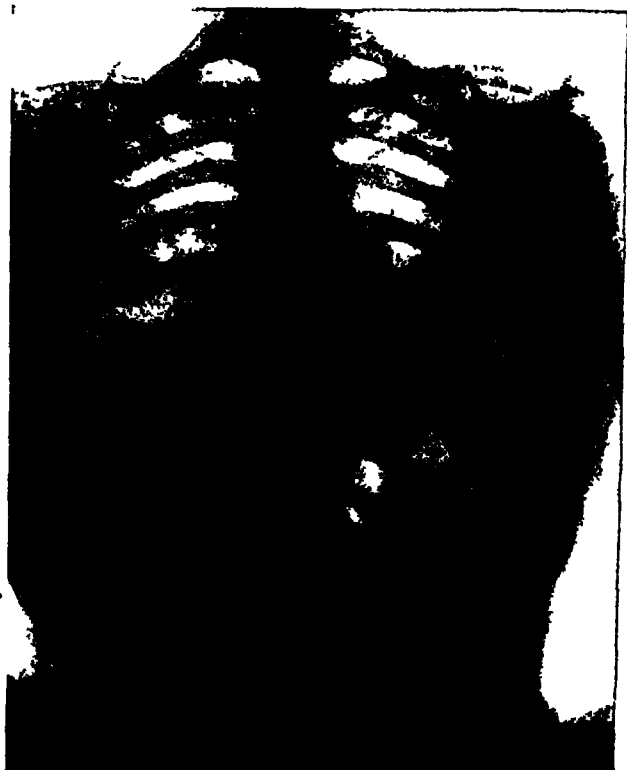


Fig 73—Case III x-Ray taken July 21 1924 A parenchymatous infiltration extends out from both root regions to the bases of the lungs The right lung is further complicated with a pleural effusion

pectoration She has had night-sweats and felt feverish in the late hours of the day She was tired easily She also complained of vague pains in her chest, especially over the sternum She lost 13 pounds in weight in the last six months Her chief complaint was cough and expectoration over a period of three months

Her temperature on admission was 100° F, pulse 104, respirations 24. Her weight was 128 pounds. She was referred from the Medical Department as a case of pulmonary tuberculosis. This diagnosis was confirmed because of the physical findings in the right upper lobe and a pleural effusion over the right base.

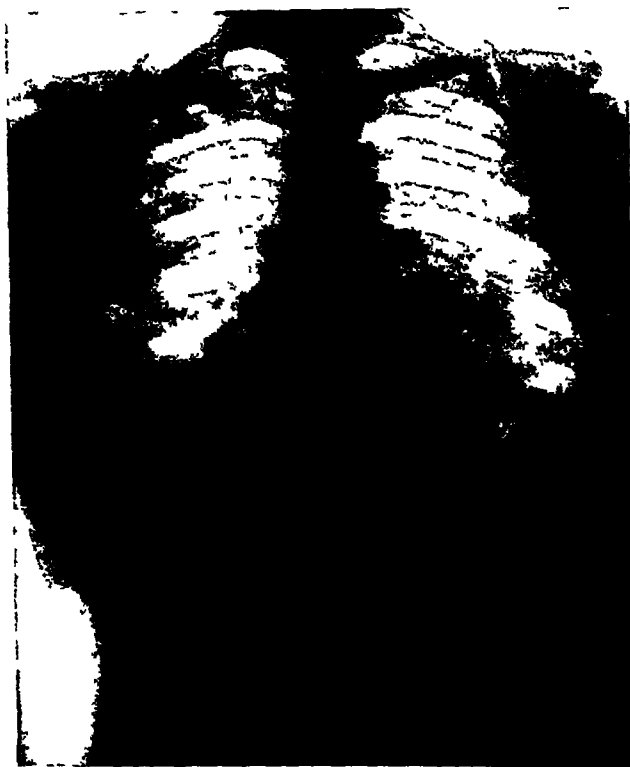


Fig 74—Case III. x-Ray taken on August 20, 1924. There is an extension of the lesion over the left base. The pleural effusion is still present.

Physical Examination—Right lung. On percussion there was flatness elicited below the ninth vertebral spine posteriorly and the fifth rib anteriorly. On auscultation bronchovesicular breathing was heard from the apex to the second rib anteriorly and to the fifth vertebral spine posteriorly. A few moist râles were heard in this area. Over the area of flatness breath sounds

were absent Posteriorly, below the eighth vertebral spine, many moist râles were heard Left lung Anteriorly, between the fourth and sixth ribs, showers of moist râles were heard

A needle was inserted into the lower right chest posteriorly and 10 c c of a clear straw-colored fluid withdrawn



Fig 75—Case III α -Ray taken September 19, 1924—sixteen days after antiluetic therapy had been instituted The pathologic shadows are beginning to clear

A diagnosis of pulmonary tuberculosis was made, complicated by a pleural effusion The patient was referred to the Vanderbilt Day Camp where she was put under the routine treatment for tuberculosis Three days later her temperature was 99.2° F, pulse 104 The findings were the same except for

a clearing up of the râles over the right apex. In the following month she was examined seven times. The physical findings never varied. Her temperature ranged between 99° and 100° F. She added 3 pounds to her weight. Her cough and expectoration were not relieved. We still thought we were dealing with

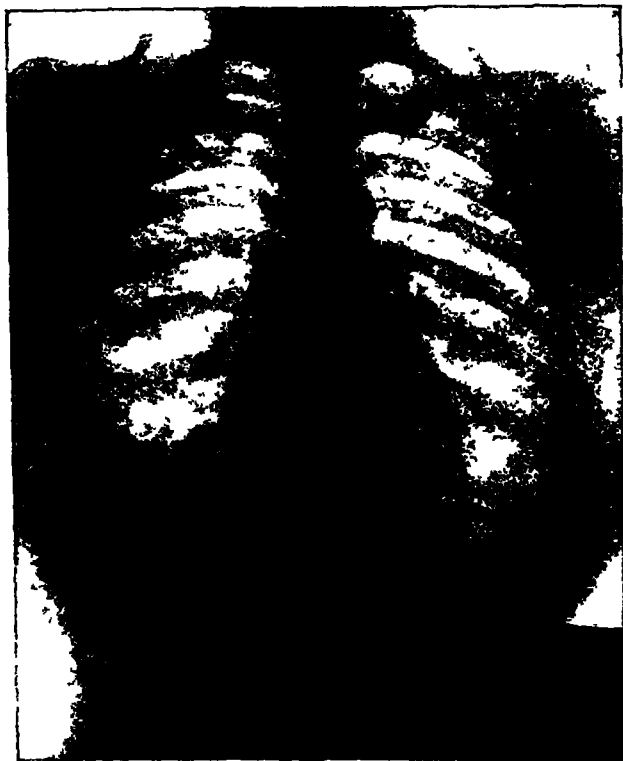


Fig 76—Case III. x-Ray taken November 19, 1924—seventy-seven days after antiluetic therapy had been instituted. There is almost a complete clearing of the lesion.

a tuberculous process. On July 21, 1924 the patient was examined again. The persistence of moist râles over the right base posteriorly suggested a bronchiectasis and a question of doubt as to a tuberculous etiology. The patient was x-rayed. The x-ray ruled out a tuberculous process. It showed an in-

filtration of the bases of both lungs with a pleural effusion over the right base. The possibility of a basilar tuberculosis was thought of. I have never seen such a condition without an apex being involved. To make sure we were not dealing with a tuberculous process, a specimen of sputum was examined each day for a week. They were all negative for tubercle bacilli.

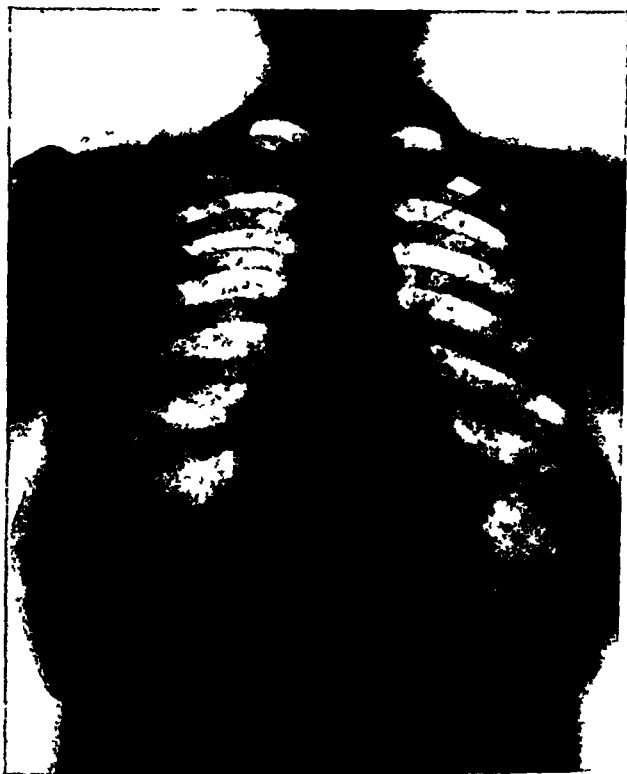


Fig 77—Case III. X-Ray taken January 6, 1925. There is no evidence of the previous lesion.

On August 11, 1924 the physical findings were unchanged. Her temperature varied between 99.6° and 100.2° F. On August 20th another X-ray was taken because of the persistent findings. This X-ray showed an increase in the markings at the left base. The density of the shadows was also increased. Very little at-

tention had been centered on the heart shadow. It suggested the possibility of an aortitis. The increase in v-ray shadows and the aortitis made us suspicious of syphilis. A Wassermann was taken and reported 4+. It was repeated and again reported 4+. The patient was placed on mixed treatment on August

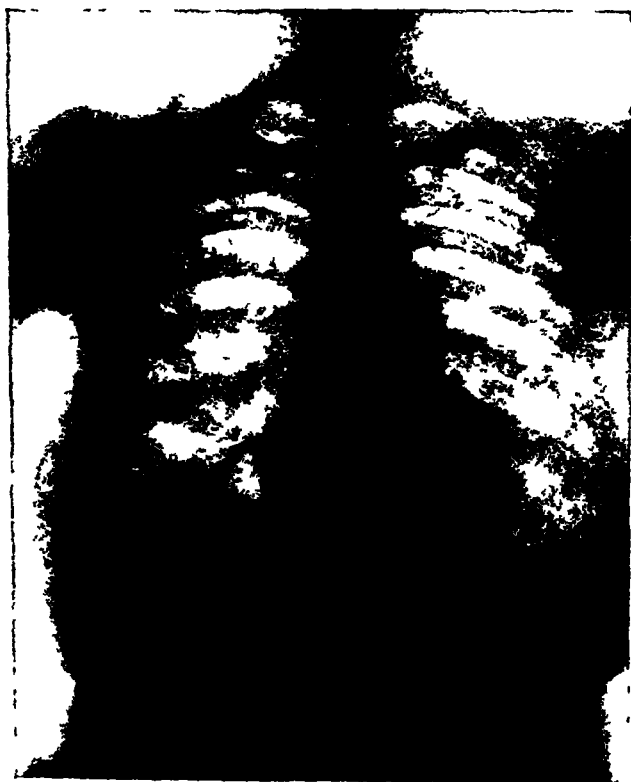


Fig 78—Case III x-Ray taken April 27, 1925. There is no evidence of the previous lesion.

27, 1924. Her temperature was 100° F, pulse 92, respirations 21, and weight 135½ pounds. On September 3, 1924 her temperature was 98.4° F, pulse 80, respirations 24, and weight 135½ pounds. On September 5, 1924 the patient was given salvarsan injections. There was an immediate improvement in the symptoms. The physical signs began slowly to disappear.

The x-rays that followed showed a gradual disappearance of the lesion. On January 1, 1925 her temperature was 98.6° F, pulse 90, respirations 24, and weight 151½ pounds. Physical examination. No modification of breath sounds or râles were heard. On June 1, 1925 her temperature was 99° F, pulse 80, respirations 24, and weight 154 pounds. Physical examination. No modification of breath sounds or râles were heard.

Discussion—This case is interesting because it was incorrectly diagnosed at first. The patient was ill for three months with cough, expectoration, fever, and all the signs of toxemia. She was then sent to the Vanderbilt Day Camp and placed on rest treatment. Under such routine treatment for three months there was no abatement in the symptoms except for a slight increase in weight. It may be argued that the patient had an intercurrent infection superimposed on a luetic condition. Such a process would not probably last six months. And, again, it may be noticed that an increase occurred in the x-ray shadows of the left lung between July 21 and August 20, 1924. The greater density in the x-ray shadows can be interpreted only as an increase in the pathologic process. It resembled closely the increase in shadows as occurs in malignancy of the lung. After the diagnosis of syphilis was made and the proper treatment instituted, not only the symptoms and physical signs but also the x-ray shadows disappeared.

It is my opinion that we were dealing here with a case of syphilis of the lung.

And from the x-ray shadows, as seen in the first two plates, I would classify it as an indurative bronchiectatic type of pulmonary syphilis.

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OBSCURE BRONCHIAL CONDITIONS CAUSED BY POLLENS

In the last few months a number of obscure lung conditions have come under my observation. They have come with diagnosis of either pulmonary tuberculosis or chronic bronchitis. The clinical picture was that of repeated sieges of acute bronchitis beginning in early spring. The attacks of cough with expectoration varied. In some cases they persisted for a time, disappeared, and then reappeared in a more exaggerated form. In other cases they were continuous. The physical signs were always scanty. There was a diminution of the intensity of the breath sounds with occasional sibilant and sonorous râles, as one would hear in an acute bronchitis. There were no other findings. The patients came to their local physicians because of a suspicion of pulmonary tuberculosis caused by the persistent cough and expectoration.

A year ago I was impressed with a case of a similar nature which developed an acute bronchitis in the month of May. This bronchitis had appeared a year previously during the terminal period of a pregnancy. With the termination of the pregnancy in the first week in August the bronchitis abruptly disappeared. It happened that the patient again became pregnant the following year with the terminal part going through the same months. In May her bronchitis reappeared. I examined her in June. There was a diminution of the breath sounds with a few sonorous râles heard bilaterally. I tested her with the pollen antigens. She reacted violently to both timothy and June grasses. In August the bronchitis ended abruptly.

With this obscure case of bronchitis in mind, and having ruled out pulmonary tuberculosis by physical and x-ray examinations, I tested a number of patients with pollen antigens. One reacted to oak and timothy grass, another to black walnut and timothy grass, a third reacted to June and timothy

grasses and ragweed, the fourth reacted to June grass. These cases did not give one the impression of asthma, and I have not included any asthmatic cases in this group.

The following case reports will give one a better impression of the clinical picture caused by some of the pollens.

REPORT OF CASES

Case I—S. B. was referred to me by Dr. Harry Cohen. The patient complained of cough and expectoration, which began in late August, 1924 and persisted to about December, 1924. He was relieved for a few months. In late April or early May his bronchitis returned. With it there was a loss of appetite and fatigue. There was a slight decrease in his weight. He had lost a sister who died of pulmonary tuberculosis, and he thought he might have a similar condition. Because of the scanty physical findings Dr. Cohen referred him to my office for an opinion. The above history was elicited. On physical examination diminished intensity of breath sounds was heard over both lungs. Sonorous and sibilant râles were heard over the upper half of the right lung. A few moist râles were heard at the right base. Stereoscopic x-ray examination revealed the following:

Right Lung—The root shadows were slightly exaggerated. The bronchial markings to the diaphragm were accentuated and along their course were seen some bronchiectatic cavities, especially in that area close to the diaphragm. There was no parenchymatous lesion.

Left Lung—The root shadows were slightly accentuated. In the eighth interspace, posteriorly, a small calcified nodule was seen—a primary tubercle which may be considered as a normal condition.

From the physical examination and x-ray a diagnosis of bronchiectasis was made, involving the right lower lobe. I became suspicious of a possible foreign body, and on questioning the patient further found that a short period previously he swallowed an apple core which became stuck in his throat and went down the "wrong pipe," as he explained. It seems possible that he might have aspirated an apple seed or a part of the

apple core, which produced these physical findings and caused this bronchiectatic condition. I suggested that a diagnostic bronchoscopic examination be made. The patient was bronchoscoped by Dr C M Myerson, who reported that he saw no foreign body.

I then tested the patient with pollen antigens. He reacted to June and timothy grasses and ragweed. On reviewing his history again it was found that his first attack of bronchitis started in August in the country where he was summering. This bronchitis lasted until the first frost. He then was well until late April when he again was seized with bronchitis. The history checks up well with the pollen antigen reactions. The first attack came with the ragweed in August and persisted way into December until the first real frost. In spring came the June grass and timothy grass pollens.

Case II—M B, female, white, thirty years of age, was referred to me by Dr I Blumenthal, of New York City. The patient had a bronchitis in February which the doctor found was secondary to an attack of a severe head cold. There was a question of the sinuses of the head being involved. *x*-Rays of the sinuses revealed no such condition. An *x*-ray examination of the chest revealed no abnormal shadows. The head cold subsided with an amelioration of the bronchitis a few weeks later. She was free of bronchitis the month of April and was congratulating herself because of her recovery when she was seized by a more exaggerated form of bronchitis. On May 22, 1925 examination of her lungs only revealed a diminution of intensity of breath sounds and occasional sonorous râles. She was tested out with the pollen antigens and reacted to June grass.

Case III—A E, female, forty-three years of age, was referred to me by Dr MacAdam. She complained of a bronchitis which started in February, 1925. She heard some snoring sounds in her chest. She had no dyspnea or asthmatic attacks. She slept through the night. In the middle of February she was

stricken with some temperature and put to bed. Her condition was diagnosed as pneumonia by a local doctor. Her temperature only persisted for two days. She remained in bed for two weeks. She complained of cough and expectoration. She frequently became hoarse, and occasionally had blood-streaked sputa. Because of this history, and substantiated by dulness, bronchovesicular breathing, and moist râles at the left upper lobe posteriorly, a diagnosis of pulmonary tuberculosis was made by Dr MacAdam. The patient was put on the usual routine rest treatment for tuberculosis. She improved, except for an increase in her cough in May. She again was troubled with her snoring sounds. Dr MacAdam heard sonorous râles over the left lung besides the findings previously mentioned. He brought the patient down to me for a confirmation of his diagnosis. On May 25th her physical findings were as follows:

Right Lung—Except for an occasional sonorous râle at the base the lung was normal.

Left Lung—Dulness was elicited from the apex to the fourth vertebral spine posteriorly and to the first rib anteriorly. Breath sounds over this area were diminished in intensity. Fine moist râles were heard posteriorly opposite the third and fifth vertebral spines. Throughout the whole lung sonorous râles were heard. A diagnosis of pulmonary tuberculosis with an acute bronchitis was made. Because of the bronchitis occurring in the early spring I tested the patient out with pollen antigens. She reacted to black walnut and timothy grass. These pollens easily explain the early spring bronchitis which complicated the pulmonary tuberculosis.

Case IV—H. H., male, white, adult, pharmacist by occupation, was referred to me by Dr M. Greenberger. The patient came to my office June 3, 1925. He had had a severe attack of coughing and expectoration through the month of April. He thought he had a bronchitis and paid no attention to it. It subsided in the early part of May. Two weeks ago he was seized with a similar attack of bronchitis. He complained of cough and expectoration. He had no sneezing or wheezing attacks. Be-

cause of the recent propaganda against tuberculosis by The New York Tuberculosis Association and because of the persistence of his cough he thought he should be examined to rule out tuberculosis. Dr. Greenberger found no evidence of any tuberculosis and referred him to me for a confirmation of his findings. On examination there was only a diminished intensity of breath sounds. No râles were heard. The x-ray examination revealed an exaggeration of the bronchial markings throughout both lungs and a questionable cylindric bronchiectasis in the right descending bronchus. I associated the early tree and late grass pollens as the etiology of the repeated attacks of bronchitis. I was rewarded with reactions to oak and timothy grass. The oak started the attacks of bronchitis in April and subsided in May, and timothy began in late May and will continue to August.

Discussion—There is no question that among men working with allergic cases this type of case is occasionally seen. Peshkin, in a personal communication, has informed me that he has recently explained an obscure case of bronchitis in a child at the Mount Sinai Hospital Clinic to sensitization to herring. He has not seen any due directly to pollens. Cooke¹ in a manuscript on hay-fever, only mentions pollen asthma as a complication of hay-fever. He expresses an opinion that persistent bronchitis often follows hay-fever due to a secondary infection of the bronchial tubes. It is interesting to also note that he mentions that all pollen asthmas are complicated with hay-fever symptoms. The above cases had no symptoms of hay-fever. Their histories and findings made one suspicious of an acute bronchitis and because of the persistence one had to rule out tuberculosis.

In conclusion, I wish only to bring attention to the possibility of pollens explaining persistent cases of bronchitis between early spring and autumn.

¹ Cooke, R. A., in Tice, Practice of Medicine, pp. 486, 510, Hagerstown, Md., W. F. Prior Company, 5, 1921.

CLINIC OF DR MALCOLM GOODRIDGE

BELLEVUE HOSPITAL

CHRONIC NEPHRITIS

THIS afternoon I am going to demonstrate 4 cases, in each of which there are good reasons for presuming that the kidneys are the seat of a chronic pathologic process. In 2 of them it is altogether likely that renal disease is primarily responsible for the clinical manifestations which obtain, while in the other 2 there is sufficient justification for the belief that the pathology in the kidneys is but a part of a general process, and the symptoms referable to the kidney are but incidents in the course of the disease.

Case I—E S, male, age seventeen years, native of Italy, eight months in United States. Admitted to the Second Medical Division of Bellevue Hospital, December 1, 1924, complaining of swelling of the feet, shortness of breath, and malaise. The history obtained on admission was as follows:

One morning early in June, on awakening, he noticed that his abdomen was distended, and that his legs and feet were swollen. One week later, these symptoms in the meantime having progressively increased, he was admitted to another institution, where it was noted that he was suffering from a generalized edema with free fluid in the peritoneal cavity.

The urine at this time contained a heavy precipitate of albumin and a few hyaline casts, and no red cells.

He eliminated 50 per cent of phenolsulphonaphthalein in two hours.

His blood-count showed red blood-cells, 5,840,000, white blood-cells, 11,800, polymorphonuclears, 70 per cent, lymphocytes, 28 per cent, and large mononuclears, 2 per cent.

On February 7th thoracentesis of the right chest recovered 2700 c c of turbid fluid Up to this time the patient had been on a diet restricted in salt, nitrogen, and fluid, without any material improvement The protein in the diet was now increased to 120 gm, fat to 60 gm, and carbohydrate to 60 gm, and 2 grains of thyroid extract twice a day was administered

February 19th the patient's general condition was much improved, the edema and the fluid in the serous sacs diminished

At the present time the patient is up and about the ward The edema has entirely disappeared There is no free fluid in the peritoneal cavity, and there is evidence of but a small amount of fluid in both chests The patient feels well The basal metabolism rate is -18 There is still a considerable amount of albumin in the urine, $3+$, and a good many hyaline casts

DISCUSSION

Edema was the earliest and most noticeable manifestation of disease It was noticed by the patient in the early morning, and was general in its distribution On his first admission to the hospital the urine was found to boil solid with albumin, but no red cells were found There was no retention of nitrogen in the blood, and but slight increase in the blood chlorids The phthalein output in two hours was approximately normal Later the blood cholesterol was found to be quite definitely increased, the serum proteins were distinctly diminished, and the globulin fraction was increased The basal metabolism was 25 per cent below normal, a matter of considerable significance, in spite of the increased surface area of the body because of edema There was fluid in both pleural cavities and in the peritoneal cavity A culture from the pleural fluid gave a pneumococcus Type II in pure culture The blood-pressure was not raised at any time during the course of the disease The eye-grounds were normal

Finally, the patient began to improve immediately he was placed on a high protein, low fat, low carbohydrate diet, together with the administration of thyroid extract, and he did not begin to improve until these particular procedures were instituted

Until recent times such a case would have been called *chronic parenchymatous nephritis*. In the Delafeld classification it would have been known as a *chronic nephritis with exudation*.

Friedrich Mueller was the first to use the term *nephrosis* to connote a degenerative lesion of the kidney, rather than an inflammatory one. Nephrosis occupies a definite position in Volhard and Fahr's classification of the nephritides. Epstein has emphasized many of the points which serve to differentiate this type of kidney lesion from the type with edema, raised blood-pressure, and disturbed kidney function. It was Epstein's theory of the cause of the edema in this type of case which induced us to place this boy on the regime which I have reviewed above.

A feature of this case which deserves especial mention is the development of a pneumococcus empyema during the course of the disease. This complication apparently developed spontaneously, at least, as far as is known, the boy had not suffered from pneumonia. Pneumococcus peritonitis has been repeatedly observed as a complication of this type of case, but, as far as I know, this is the only case in which a pneumococcus empyema developed.

The prognosis for this boy is relatively good. We shall continue the high protein diet until the albuminuria clears up and until the serum proteins reach more nearly normal values. The thyroid administration will be continued until the basal metabolism approaches normal.

Relapses are prone to occur, a fact which should stimulate careful observation for a period of years after apparent cure.

The condition is not a common one, indeed, it is the rarest of all the forms of renal disease which I shall present this afternoon. Little is known as to its etiology.

Case II.—R. S., age sixty-five years. For many years this patient has suffered from a low-grade colon bacillus pyelitis, which was extremely resistant to treatment. The symptoms of the infection were an occasional chill followed by fever, frequent and painful micturition, and hematuria.

Since 1913 repeated urinalyses showed a trace to a marked trace of albumin. Kidney function tests performed at intervals during the period from 1913 to 1924 demonstrated but slight variations from the normal. The blood-pressure was normal excepting on one occasion, March 22, 1923, when it was 190/90, a month later the blood-pressure was 140/80. The Wassermann was negative. x -Rays of the kidney showed these organs to be large, but otherwise the kidney shadows were normal.

On February 2, 1924 he was given 6 mg/kg mercurochrome—220 intravenously. There was considerable reaction to the drug, he vomited twice, there was slight salivation and some diarrhea. However, five days later there was but a trace of albumin in the urine and a few bacilli, and the patient felt quite well in every way. He was discharged from the hospital, definitely improved, on February 12, 1924.

The latter part of February he caught a severe cold, which he neglected. It was not until nearly three weeks later, March, 1924, that he reported for observation. On the morning of admission he discovered, on awakening, that his eyes were swollen and his face was puffy. For two weeks prior to this date he had noticed that he was gaining rapidly in weight and that his urine had become scanty in amount.

On physical examination the most striking finding was the pallor of the skin and the marked, generalized, pitting edema. There was no fluid in the serous sacs, the heart was not hypertrophied, the sounds were of fair quality, the rhythm regular, the rate 80 to the minute, the peripheral vessels were not perceptibly thickened, the lungs were clear, the liver was not enlarged. The blood-pressure was 160/90, temperature, 98.6° F.

The laboratory findings on March 19, 1924 were as follows. Urine. Output in twenty-four hours, 1750 cc, specific gravity, 1.012, albumin, massive precipitate, 11 gm per L, chlorids, 4.9 gm in twenty-four hours. Microscopically, there were many pus-cells, mostly clumped, no red blood-cells, no casts. Phenolsulphonephthalein test. First hour, a trace, second hour, 9 per cent. Blood chemistry. Non-protein nitrogen, 53 mgm, uric acid 5.3 mgm, creatinin, 2.6 mgm, blood chlorids, 537.5 mgm per 100 c.c.

The patient was put to bed on a low nitrogen low salt, low fluid régime. There was an almost immediate improvement in both the clinical symptoms and the urinary findings. The edema diminished to a perceptible degree. The urinary output rose to 2000 c.c. on a 1000 c.c. intake, the chlorids in the urine rose to 8 gm. per L. on an intake of not more than 1 gm., the amount of albumin fell to 6.5 gm. per L. The blood chemistry remained practically unchanged, except for a fall in blood chlorids to 450 mgm.

This improvement was progressive until early in April, when the patient complained of feeling weak. The pulse-rate rose, the blood-pressure fell to 106/90, a gallop rhythm developed at the apex, the urinary output diminished, and the edema, which had never entirely disappeared, increased. The non-protein nitrogen rose to 68.9 mgm., the creatinin to 3.49 mgm., the uric acid to 5.6 mgm., and the blood chlorids to 500 mgm. per 100 c.c.

Massive doses of digitalis were administered, and theocin, 3 gr., three times a day, was given for one day. Under digitalis therapy the blood-pressure rose to 128/80, the pulse-rate fell to 72, the urinary output increased, and the edema diminished.

From this time until January, 1925, there was a progressive rise in blood-pressure, an increase in urinary output, a decrease in the edema up to the point of complete disappearance. At the same time there was a decreased ability on the part of the kidney to concentrate urine, and a steady rise in the blood nitrogen.

In January, 1925, the patient began to complain of nausea and headache. In February, while eating dinner, he had an attack during which he was unable to use his right hand and was unable to speak. This seizure lasted but a few minutes and then cleared up, without residual paralysis or aphasia. The blood-pressure at this time was 220/110, the pulse 96, on examining the eye-grounds the retinal arteries were found moderately sclerosed, but there were no retinal hemorrhages or exudate.

Since January the patient has continued to complain of nausea at times, there has been some headache and some twitching of the muscles. The blood-pressure has remained constantly above 200.

The blood chemistry, done April 2d, revealed a non-protein

nitrogen of 86 mgm , uric acid 84 mgm , and creatinin 8 mgm The phthalein output was not enough to read in two hours The urine contained 4+ albumin, the specific gravity varied between 1 005 and 1 009, the night urine measured 720 cc The CO² combining power was normal

At the present time the blood chemistry is as follows Urea N 80 mgm , NPN 133 mgm , uric acid 91 mgm , sugar 1666 mgm , creatinin 82 mgm , chlorids 5625 mgm The blood-pressure is 240/150, and yet the patient has few complaints, there is no albuminuric retinitis and, as you see, he does not look especially ill

DISCUSSION

The chronic infection of the genito-urinary tract was undoubtedly a predisposing factor in the sequence of events which followed soon after the administration of mercurochrome It would be natural enough to suspect that mercurochrome was the exciting factor in producing the clinical picture which occurred after its administration Mercury, if administered in sufficient amounts, causes necrosis of the tubular epithelium, and, if the kidney recovers at all from the lesions caused by the drug, the recovery is usually complete It is not at all likely that if the mercurochrome had had any effect in producing subsequent events, the patient could have been discharged from the hospital ten days after its administration, feeling quite well in every way, and with less albumin in his urine than had obtained on his admission to the hospital

Inasmuch as, at times at least, infection bears a definite etiologic relationship to nephritis, and inasmuch as this patient's symptoms dated from early in March, shortly after having contracted a "severe cold," it seems to me more than likely that the infection was the immediate cause of the change in the clinical course of the disease which took place at that time

The clinical picture in this case was quite different from that which obtained in Type I Both cases were associated with edema, but in this instance there was a moderate increase in blood-pressure, a marked reduction in ability to put out phthalein, and moderate nitrogen retention In other words, the edema in

this case was associated with very definite disturbance in kidney function. Improvement occurred when the intake of fluids, salt, and nitrogen was restricted. As the edema diminished the urinary output rose considerably. At the same time the chlorids in the urine rose in spite of a very low salt intake; the amount of albumin fell, and the blood chlorids returned to normal values. Cardiac decompensation, occurring early in the course of the disease, was readily controlled by full doses of digitalis.

The clinical picture has changed completely in the past six months. We now see a subject with no edema, with more marked hypertension (blood-pressure 220/120), quite marked retention of nitrogen, and no phthalein eliminated in a two-hour period. The ability to concentrate urine is diminished, the night urine is increased.

This case illustrates very well one of the difficulties which one meets with in attempting to group the nephritides, even from the clinical standpoint. Early in the course of the disease the prominent features in this case were edema, massive albuminuria, and moderate hypertension, while later the absence of edema was a conspicuous feature of the clinical picture.

The fact that at the present time the patient feels and looks well and has slight if any uremic manifestations, should not mislead us into predicting a favorable outcome. Such marked impairment of kidney function in chronic nephritis almost invariably spells early disaster, with death in uremia.

Patient died in uremia July 2d

Case III — N. S., age forty-two years. Fifteen months ago the patient began to be disturbed by nausea in the early morning, and was troubled with persistent pruritus. He had some headache on awakening, never severe. The nausea and headache lasted one or two hours after breakfast, and then disappeared. At about the same time he began to lose in weight, he thinks he has lost about 30 pounds since the onset of his symptoms.

During the past year he has noticed a marked increase in the daily quantity of urine passed, and he has had nocturia three or four times.

Four months ago he began to suffer from severe cramps in the muscles of his legs, especially at night, and more recently he has complained of severe pains in both lower extremities, particularly on climbing stairs. These pains disappear completely after a few minutes' rest.

Lately he has been troubled with vertigo, and has become more irritable.

Past history Does not remember any serious illnesses. He has never been susceptible to infection. He denies lues and gonorrhea.

On physical examination, at the time of admission, the patient looked ill, he had evidently lost in weight, his skin was pale and there were irregular areas of pigmentation over the lower extremities, shoulders, chest, and back. There was moderate exophthalmos, the left pupil was dilated and reacted promptly to light, the sight in the right eye had been destroyed forty years before by an accident, the left fundus showed a beginning neuroretinitis. The mucous membranes were pale, with no pigmentation. The tongue was heavily coated and tremulous. The lungs were normal. The heart was hypertrophied, the sounds of fair quality, with no murmurs, the rhythm was regular, the rate 80 per minute. The peripheral vessels were distinctly thickened, the blood-pressure was 210/110. The abdomen was negative, except for a right indirect inguinal hernia. The prostate was normal. The reflexes were everywhere increased.

The laboratory findings on admission were as follows. **Urine** Specific gravity 1010, albumin, a heavy trace, microscopically, a few red cells, no casts. Daily output of urine, 2000 c c, night specimen, 800 c c. Phenolsulphonephthalein test. First hour, no trace, second hour, a faint trace. **Blood-count** 2,696,000 red blood-cells, hemoglobin 58 per cent, 6200 white blood-cells, differential count normal. **Blood chemistry** Uric acid, 5.7 mgm, creatinin, 9.9 mgm, urea nitrogen, 61.6 mgm, non-protein nitrogen, 140 mgm, sugar, 182 mgm, and chlorids, 496 mgm, per 100 c c blood.

Two months ago a venesection was performed, followed by transfusion, with temporary improvement in symptoms. Since

his admission to the hospital the diet has been restricted in nitrogen and salt. The fluid intake has varied between 1000 and 2500 c c. The emunctories have been encouraged by the use of cathartics, colon irrigations and hot packs.

In spite of treatment the patient's condition has grown progressively worse, the headache is now severe, there is persistent nausea, with vomiting, the patient is at times delirious, there is a distinct uremic odor to his breath, there is an albuminuric retinitis in the left eye, with choking of the disk. The blood-pressure today is 260/155. The patient is dyspneic. The urea nitrogen has risen to 110 mgm., and the death of the patient can be only a question of a few days.

DISCUSSION

You will note that the clinical features of this case are quite different from those which obtain in Case I, and in the early phase of Case II. The patient has never suffered from any serious illness, and has never been susceptible to infections.

The nausea and headache, which were the first symptoms complained of, were no doubt uremic manifestations, which means that the kidney pathology had reached an advanced stage in its development before the patient realized that he was ill. Once symptoms had appeared, there were no periods of remission. On the contrary, the clinical course of the disease was steadily progressive. At no time has there been any edema.

The look of the patient is altogether different from that in either of the other cases presented. There has quite evidently been a considerable loss in weight, so that the appearance is that which one observes in one of the wasting diseases.

The uremic manifestations, the changes in the eye-grounds, and the very marked impairment of kidney function enables us to predict with assurance that a fatal issue is impending.

Case IV—T. H., age fifty-two years. Case history. The patient was first seen on September 23, 1924. On arising the morning of that date, he complained that his "throat felt queer," and his tongue seemed swollen. He had difficulty in talking

His wife noticed that he had considerable difficulty in pronouncing his s's. He did not note any weakness of either arm or leg, but, while shaving, he discovered that his face was "drawn somewhat to the left."

For years he had worked under high tension, and for a week prior to his present attack he had been under unusual mental strain.

Past history. He has never suffered from any serious illness, in fact, he has always been unusually well. More recently he has been very nervous and at times greatly depressed and easily fatigued. A few years ago he had been told by a physician that his blood-pressure was somewhat above normal.

He used alcohol and tobacco regularly, but in moderation. He had a good appetite and indulged it, in consequence of which he had put on considerable weight in the past five years.

Physical examination. The patient did not look acutely ill. He was overweight, 234 pounds stripped. Height 6 feet, 1 inch. He was exceedingly depressed and emotionally unstable, crying two or three times during the examination. There was perceptible paresis of the right side of the face, the mouth was drawn slightly to the left, the tongue protruded straight. The pupils were equal and reacted to light, there was distinct sclerosis of the retinal arteries, with increase in the central light reflex and compression of the veins at the crossings, there was a small hemorrhage in the region of the left macula. The heart was hypertrophied, the first sound at the apex was of poor quality, the second sound reduplicated, there were numerous premature systoles, a soft systolic murmur was heard at the base in the second right interspace, the second aortic sound was accentuated, the pulse-rate was 86 to the minute. The peripheral arteries were distinctly thickened, the blood-pressure was 196/126. The lungs were clear. The abdomen was quite negative. The tendon reflexes were moderately increased on the right side, there were no pathologic reflexes.

The urine contained a faint trace of albumin, and no casts, the specific gravity varied between 1026 and 1034, the quantity of night urine was normal. The phthalein output was 20 per cent the first hour, 21 per cent the second. The blood-count

showed 5,300,000 red blood-cells, hemoglobin 92 per cent., 5500 white blood-cells, the differential count was normal. The blood chemistry showed a non-protein nitrogen of 32.9 mgm, uric acid, 4.7 mgm, creatinin, 1.5 mgm, and sugar, 97 mgm per 100 c.c.

Treatment The patient was kept in bed for one month, and then allowed up for a few hours, each day, for another month. His diet was reduced to about 1800 calories a day, the proteins in the diet were reduced to 70 to 80 gm daily, salt was reduced to the quantity cooked with food, fluids were permitted in sufficient quantity to relieve thirst. Alcohol and tobacco were forbidden. The bowels were kept freely open. When he returned to work he was cautioned to reduce his working hours materially. He was warned against physical and mental strain. This régime has been followed conscientiously up to the present time.

The paresis of the face cleared up in a few days. The retinal hemorrhage was absorbed in a month's time. There have been no subsequent cerebral manifestations. Today his weight is 185 pounds, a reduction of nearly 50 pounds in body weight. The blood-pressure is 168/108.

The urine contains no albumin and is of normal specific gravity, 1018 to 1026. The blood chemistry shows a non-protein nitrogen of 25 mgm, uric acid 4 mgm, and creatinin 1.2 mgm per 100 c.c.

Subjectively, in his words, "I am feeling fit, quite like my old self."

DISCUSSION

If you contrast Case IV with any of the three preceding cases, you will note marked differences in the history, the mode of onset, the patient's general appearance, and the study of kidney function.

The first indication that he had of anything wrong in his cardiovascular system was the attack of dysarthria, with right-sided facial weakness, which occurred in September, 1924. Prior to that time, he had had symptoms which might have been mistaken for some functional nervous disturbance, such as so-called neurasthenia.

You will note that the patient is a large, well-nourished man,

in spite of the fact that he has lost nearly 50 pounds in weight in eight months. He has moderate cardiac hypertrophy. His blood-pressure is 168/108 at the present time, six months ago it was 196/126. There has never been any retention of nitrogen, excepting a slight increase in his blood uric acid. There has been good ability to concentrate urine, the specific gravity has been as high as 1034 on one occasion, when fluids were considerably restricted, in other words, when subjected to strain. His phthalin output has been slightly below normal, 41 per cent in two hours. His response to régime has resulted in a fall in blood-pressure and an improvement in his subjective symptoms, and altogether he presents quite a different picture from that which obtains in Cases II and III, which were also associated with hypertension.

Such cerebral attacks as he had in September have been explained in various ways, so-called intermittent claudication, spasm of the branch of the midcerebral which supplies the internal capsule, areas of localized edema, etc. It seems to me that the most satisfactory explanation offered for the attacks of aphasia and the monoplegias which occur with this disease is that they are due to small hemorrhages widely scattered in the cortex of the brain.

The terminal stage in this type of case is rarely uremia. It is more likely that it will run a long course, and that the end will be brought about by a cerebral accident or failure in cardiac compensation or intercurrent disease.

The differences between this type and the one immediately preceding are obvious. Case III represents a chronic nephritis with hypertension and failing kidney function, while Case IV represents a case of hypertension with good kidney function. The progress of one is comparatively rapid and ends in uremia, while, in the other case, the disease is very slowly progressive, and the terminal factor is usually either cardiac failure or a cerebral accident, and very rarely uremia.

CONCLUSIONS

Since Bright's original thesis, published in Guy's Hospital Reports nearly one hundred years ago, classifications of nephritis

have been beset with many difficulties. Pathologic classifications have been legion. Kidneys have been grouped according to their gross and histologic appearance, but, unfortunately, it is difficult to correlate the clinical manifestations of nephritis, on the one hand, and structural changes, on the other. We are not able to predict that a given case of nephritis will furnish at autopsy a kidney with a definite pathologic picture, either in terms of its size, its color, or its histology, nor is there any close parallelism between kidney function and kidney structure.

It has been my experience, in teaching, to find that the attempt to detach certain types of cases in which the kidney structure is certainly concerned from a clinical classification of nephritis, and to substitute such terms as *nephrosis* and *essential hypertension* has had the effect of still further confusing a problem which was already difficult for the student of medicine to comprehend.

Clinically, one sees two types of renal disease with wide-spread edema, one with raised blood-pressure and one without, and two types of cases with hypertension without generalized edema, one with kidney function more or less impaired and the other with little or no depression of kidney function.

These four clinical types are pretty well represented in the cases which you have seen this afternoon.

That one should see a case of chronic nephritis with edema and moderate increase in blood-pressure at one time, and at some later period should observe this same case free from edema but with marked increase in blood-pressure and failing kidney function, as happened in Case II, in no way changes the value of a simple classification. It is not only possible for one type of chronic nephritis with edema to change to a chronic nephritis with hypertension without edema, and failing kidney function, but it is also possible for a case of hypertension with good kidney function, the benign type of the Volhard and Fahr classification, to terminate in uremia with all the symptoms incident to the terminal stage of the diffuse glomerulonephritis of this classification. This has actually happened in about 5 per cent of the cases in my records.

The variables which occur in the clinical course of chronic nephritis add greatly to the difficulties which surround the proper understanding of the subject, and it must continually be borne in mind that, while pure types of chronic renal disease occur in a large percentage of cases, very puzzling admixtures of types also take place

It is when one comes to consider prognosis that the importance of recognizing and differentiating clinical types of chronic renal disease is especially emphasized. With a little experience you will be able to predict with some degree of accuracy the immediate and ultimate prospect in a fair number of the cases under your observation, and you will find that you are basing your prophecy very largely on a study of kidney function, together with the presence or absence of high blood-pressure

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RHEUMATIC HEART DISEASE

AN understanding of rheumatic heart disease demands a thorough knowledge of the nature and course of the rheumatic infections. Cardiac involvement is an incident in the course of these infections, and the progress and development of the cardiac lesion depends in large part on the nature of the rheumatic process.

History.—As early as 1788 David Pitcairn noted the occurrence of rheumatism and heart disease in the same individual. In 1809 Dundas published clinical and pathologic findings of a number of cases of heart disease in the young, which followed one or more attacks of rheumatism. In one instance he observed the cardiac disorder develop during the acute rheumatic episode. He believed that in these cases the rheumatism had attacked the heart. In a paper entitled *On Rheumatism of the Heart*, Wells, in 1810, pointed out that rheumatism and heart disease were often associated in the same individual, and drew the conclusion that these two diseases depend on a common cause. Hope, in 1832, made the statement that inflammation of the heart or its membranes is a frequent and formidable complication of acute rheumatism, but it was Bouillaud, who, in 1833, was the first strongly to emphasize the importance of this relationship. Indeed, he postulated the following law: In acute, generalized, febrile articular rheumatism the coincidence of an endocarditis, or pericarditis, or endopericarditis is the rule, the non-coincidence the exception. Ten years later Todd made a further contribution to our understanding of rheumatic heart disease when he wrote, "I have now so frequently met with instances of diseased heart in young persons, not traceable to an actual paroxysm of

rheumatic fever, who, nevertheless, showed evident marks of a rheumatic diathesis which had existed for a longer or shorter time, that I cannot but regard this state of constitution as a fertile source of those cardiac diseases which are met with in early life " By rheumatic diathesis he meant the vague muscle and joint pains without swelling, with or without fever or anemia. He also pointed out the close relationship between chorea and heart disease and the rheumatic state



Fig 79 —Rheumatic endocarditis, mitral valve

Etiology—Rheumatic fever is an acute infectious disease of unknown etiology, common in childhood and up to the fourth decade of life, but rather infrequent in the later age periods. Some authors have sought to prove that a streptococcus is the specific organism of the disease, but the evidence for this is inconclusive. Rheumatic heart disease is a little more common in

females than in males Of 1036 cases collected by Coombs, 64 per cent. were women Of 199 consecutive cases at Montefiore Hospital, 55 per cent. were females Environment plays a significant rôle in the incidence of the disease Rheumatism with its sequelæ is a disease of temperate climates, more common in cities than in the country Poor housing seems to favor its development On a number of occasions I have seen children with rheumatic heart disease who remained well and free from reinfection during a stay of many months in a convalescent home, suffer a new attack of chorea or tonsillitis within a few days after their return home In view of the fact that there was very little rheumatism among soldiers on the battlefields of France, exposure to wet and cold does not seem to play a vital rôle in predisposing to rheumatic infection The seasonal variations of rheumatic fever are well known In the eastern United States the highest incidence of cases is in February, March, and April, in London, September and October show the greatest number of cases

It is generally assumed that the tonsils are the chief portal of entry for the rheumatic virus While this is apparently true in many instances, too much emphasis has undoubtedly been laid on this view The fact that tonsillectomy does not prevent rheumatic reinfection is strong evidence against it It is safer to regard the whole respiratory tract, but particularly its upper portions as the avenue of entry of the virus

Heredity undoubtedly plays an important rôle in the distribution of rheumatic fever and, consequently of rheumatic heart disease Cheadle studied the family histories of 592 children admitted to the hospital for all kinds of diseases Of 173 who gave a clear history of acute rheumatism in immediate blood relatives, 38, or 20 per cent, had unmistakable rheumatic infections, while of 139 with negative family histories, only 15, or 4.5 per cent, had had articular rheumatism

The rheumatic infection may manifest itself in a variety of clinical pictures Among the most common of these are tonsillitis, pharyngitis, chorea, acute articular rheumatism, carditis, erythema multiforme, pleurisy, rheumatic nodules and myositis

or "muscular rheumatism" Characteristic of the disease is the recurrence of the infectious process, as well as the multiplicity of its manifestations. This is particularly well marked in children, in whom attacks of tonsillitis may alternate with chorea, arthritis, carditis, and the various cutaneous manifestations. In childhood arthritis is less pronounced and less severe, but in adults the virus is usually localized in the joints and in the heart.

As Barlow has aptly said, "The tendency to recurrence of rheumatism is almost as marked a feature as that to relapse, and the after-attacks of rheumatism are as worthy of our consideration as the relapses. How often these consist of a day or two of inconvenience only, with slight effusion into a joint or the structures round a joint, or some vague muscular pains! In the cases in which the heart has suffered we are apt to think of the patient's subsequent progress as depending on the more mechanical considerations or cardiac compensation. But in many of the febrile attacks of old rheumatic heart cases the solitary insidious intercalation of just one small joint effusion may remind us that we have once again to deal with a rheumatic fever, the principal incidence of which is in valves or pericardium."

In certain cases, particularly in children, the infection is very insidious in its onset and in its progress. Anemia, malnutrition, nervousness, and irritability with fleeting muscle and joint pains may be the only manifestations until a careful physical examination reveals organic changes in the heart.

The course of the disease varies. Some few cases run an acute virulent course and terminate in a few weeks from hyperpyrexia or cerebral rheumatism, or from a massive cardiac infection. In most instances the period of active infection is from two to six weeks. There remains, however, the great probability of recurrence. In other cases, again, and these are seen particularly in children, the infection at no time appears to be of great intensity, yet the disease drags on for weeks and months, one manifestation following the other. Tonsillitis, joint pains, cardiac symptoms, pleurisy, cutaneous eruptions may follow one another in slow procession over a long period of time.

The important fact to bear in mind is that in every case of

infection with the rheumatic virus there is grave danger of cardiac infection with permanent damage to its muscle or to its valves. The more persistent and stubborn the infection, the greater the danger, the more frequent the recurrences, the greater the probability that the heart will be involved. It is the continued and repeated infections rather than the acutely severe ones that endanger the heart. For instance, there is no relationship between the severity of the tonsillitis and the arthritis and the likelihood of the localization of the virus in the heart.

The cardiac symptomatology may suggest specifically an endocarditis, pericarditis, or myocarditis, but in most instances a diagnosis limited to one of these three categories considers only the presenting symptom. When the heart is implicated, the endocardium, pericardium, and myocardium all are involved to some extent; the condition in reality, is a carditis. In children involvement of the heart is particularly frequent. From 80 to 90 per cent of children under ten years of age, who have passed through a rheumatic infection, present signs of cardiac disorder.

The time between the onset of infection and the appearance of signs of cardiac involvement is a variable one. At times the heart appears to be the first organ affected. Often evidence that the rheumatic virus has localized in the heart is not apparent until some months after the onset of the first rheumatic invasion. Some individuals pass through a number of attacks of chorea, or articular rheumatism, before their heart is attacked, in others, and these, unfortunately, are in the minority, this vital organ is permanently spared.

It is difficult to find in the literature accurate figures of the frequency of cardiac involvement in the rheumatic infections. The statistics of different authors show considerable discrepancies. The younger the patient, the more probable the development of rheumatic heart disease. In children under ten the incidence is probably over 80 per cent. In older individuals at least 50 per cent. of rheumatic infections eventually are complicated with heart disease.

The recognition of cardiac infection in rheumatic fever depends in part on the exhaustiveness of the diagnostic methods

employed Unless recourse is had to instrumental studies many cases will be overlooked

Cohn and Swift, in an intensive study in which daily electrocardiograms were taken of cases of rheumatic fever, found that the heart was affected in 35 of the 37 patients in their series The evidence was of three sorts an increase in the duration of auriculoventricular conduction, alteration in the ventricular complex of the electrocardiogram, and the occurrence of numerous irregularities of rhythm In most cases these changes in the electrocardiogram were transient

The so-called rheumatic nodules, originally described in 1868 by Hiller, and in 1875 by Meynet, when present, are pathognomonic of rheumatic infection They may vary from 1 mm to over a centimeter in diameter and are always related to deep fascia, tendon, or periosteum, they are subcutaneous and the skin is freely movable over them The smaller ones are more or less movable on the deeper parts and are more often connected with tendons and fasciæ The larger nodules are more frequently fixed to the periosteum Microscopically, they are seen to consist of fibrin, fibroblasts, and accumulations of large round-cells somewhat resembling the Aschoff bodies in the myocardium They are found most frequently over the olecranon process, about the malleoli and patellæ, over the spinous processes of the vertebræ, along the superior curved line of the occiput, and on the extensor tendons of the forearms The appearance of the nodules is always associated with an active rheumatic infection Commonly they develop in successive crops They may persist for only a few days, or may remain for many months In 1 case that came under my observation they had been in existence for over six months They are not painful Rheumatic nodules are common in children, but rare in adults Their significance lies chiefly in the fact that in the majority of cases they are associated with a grave heart lesion In a child who presents nodules we expect to find a severely damaged heart It is claimed that their presence indicates an unfavorable prognosis This has not been invariably so in my experience There is no doubt, however, that in the majority of cases they are an unfavorable

RHEUMATIC HEART DISEASE

activity of the rheumatic virus. Old nodules may persist for years, even, long after the infection has ceased. This is well illustrated by the following 2 cases.

Case I—A boy, ten years of age, was admitted to the Hospital with a history of chorea, followed by rheumatism two months previously. During the course of his illness appearance of nodules all over his body was noted. At



Fig S0 —Rheumatic nodules on arm and occiput

of examination he was afebrile and presented signs of ²⁶⁸ sufficiency and mitral stenosis with no evidence of ²⁶⁹ of the circulation. Nodules varying in size from 4 ²⁷⁰ cm were very evident on the head along the upper ²⁷¹ ridge, and on the parietal bones, along the spinous ²⁷² the vertebræ, at the olecranon processes and condyles ²⁷³ humeri, attached to the periosteum on the outer ²⁷⁴ radius, along the extensor tendons of the hand and ²⁷⁵ around both malleoli and patellæ.

Case II—Another boy, eight years old, who was admitted with a subacute pericarditis with effusion and mitral stenosis, had large nodules about both patellæ and olecranon. Six months later, although they had receded in size, they were still quite evident. The patient had recovered from his acute symptoms and was up and about in good condition.

In cases in which the evolution of a heart lesion is observed during the course of one of the rheumatic infections, or in patients exhibiting organic cardiac defects in whom there is a clear history of an infection with the rheumatic virus, it is a simple matter on empiric grounds to relate the heart lesion to the accompanying or antecedent infection and to make a diagnosis of rheumatic heart disease.

There are many individuals, however, in whom the clinical signs and, indeed, the pathologic changes in the heart resemble in every respect those characteristic of rheumatic heart disease, yet who, as far as can be ascertained from their history, have never had any of the infections of the rheumatic cycle. It is in these cases that it is impossible to make an accurate etiologic diagnosis. The majority of them, particularly in the younger age groups, undoubtedly belong to the rheumatic category and must be classed as such until the time when the nature of the specific rheumatic virus is discovered and methods are developed for its clinical recognition. The milder and aberrant rheumatic manifestations, such as pharyngitis, muscle pains, or erythemas, are so readily overlooked both by doctor and patient that it is not strange that a rheumatic history is so often missed in these individuals. On the other hand, cases do occur in which an endocarditis or myocarditis resembling that induced by the rheumatic virus develops consequent on other infections, such as scarlet fever, typhoid fever, and pneumonia. Occasionally a chronic osteomyelitis is followed by progressive valvular heart disease. Bronchopneumonia, particularly the type seen following influenza, at times is complicated by the development of arthritis and simple myocarditis and endocarditis.

A young man was stricken with a mild bronchopneumonia following influenza. After the temperature had defervesced

the wrist- and ankle-joints became swollen and red and there was a recrudescence of the fever. A few days later, with a continuance of the fever, the arthritis subsided, but the pulse became rapid and a systolic apical murmur became audible. The following day the heart became very irregular due to a perfect jumble of extrasystoles. In about ten days the signs of active infection and the cardiac irregularity subsided, but slight enlargement of the heart and the murmur persisted.

The problem of the relationship of rheumatic fever to heart disease may be studied from another angle. Over 50 per cent. of all cases of chronic heart disease, from a study of their histories, can be classed as rheumatic. That means that a still higher percentage of those suspected of rheumatic heart disease will give a positive history.

Because of our ignorance as to the nature of the rheumatic virus the classification at best is unsatisfactory. On the one hand, there are the many cases in which it is impossible to determine any particular etiologic agent, and which resemble in their course and in their pathology those which are definitely rheumatic. On the other hand, if rigorous pathologic criteria are applied and only those cases classed as rheumatic which exhibit Aschoff bodies on postmortem examination, the number of positive cases will be still fewer. Libman found Aschoff nodules in only 18 out of 56 cases of clinically definite rheumatic heart disease. Again the point must be emphasized that the rheumatic infection is often so insidious that it may escape recognition, a fact which will account for the negative history in many cases.

In childhood and in early adult life rheumatic fever is the chief cause of heart disease. The extent of its ravages is shown by several statistics. Physical examination of children in New York City schools revealed about 1.5 per cent. with cardiac defects. About 2 per cent. of the young men examined for enlistment in the U. S. Army during the recent war were rejected because of heart lesions.

Pathology—Infection of the heart with the rheumatic virus results in a pancarditis. With rare exceptions it is incorrect to speak of rheumatic endocarditis or pericarditis as distinct en-

tities, for they are almost invariably associated with a myocarditis as well. The lesions of rheumatic myocarditis are quite characteristic and in certain stages of their development are pathognomonic. The specific lesions, which are microscopic in size, were first described by Aschoff in 1904, and are known as Aschoff bodies, or submiliary nodules of rheumatic fever. They occur near the small or medium sized vessels, usually in close relation to the adventitia, and consist of large cells with one or more large polymorphous nuclei. The cells are commonly arranged in the form of a fan or rosette and stain characteristically with pyronin-methyl-green. Lymphocytes and polymorphonuclear leukocytes are also found in the nodules. Aschoff bodies are found most frequently in the walls of the left ventricle and only in patients who have experienced a recent rheumatic infection. In the course of time they undergo central necrosis with following fibrosis and the characteristic cells disappear. In cases, therefore, in which the infection antedates death by a long period only sclerotic patches remain as evidence of the preceding myocarditis. The lesions cause injury to the heart by direct effect on the myocardium, if they are numerous, or by interference with the conduction of the cardiac impulse when they happen to involve the more important segments of the conducting system. They are responsible for the appearance of heart-block in these patients.

Acute rheumatic or verrucous endocarditis is characterized by small, grayish-white, smooth and glistening sessile vegetations inserted along the line of closure of the valve leaflets. They usually occur in a beaded row, and in a given attack all appear to be of the same age. At an early stage of their development they give a sandpaper-like appearance to the valve. When larger they resemble small warts, hence the name "verrucae," and attain a diameter of 2 to 3 mm. Often they become fused. Microscopic examination shows that the vegetations are composed of an organizing hyaline thrombus. The valve at the base of the thrombus, and at times the vegetation itself, is infiltrated with mononuclear and polymorphonuclear leukocytes. The endothelium is seen growing up over the thrombus. At times bac-

teria may be cultured from the lesions. They are, however, accidental secondary invaders, since they give rise to no immune bodies in the patient's serum. Because of the fact that the vegetations are non-friable and firmly attached to the base of the valve, emboli are never released in the blood-stream. The end-result of this process is fibrosis and thickening of the valve leaflet. In some instances the calcium is deposited directly in the hyalinized vegetation. Yet it is a far cry from such a simple thickening of the valve flap to the marked valvular deformities observed in long-standing cases of rheumatic heart disease. Here the leaflets become tremendously thickened, fibrous and distorted, adjacent valve flaps become fused, the chordæ tendinæ become shortened, and calcareous deposits of varying degrees become incorporated in the scarred valve. Such is the appearance of the chronic valvular defects. The left, more rarely the right, auriculoventricular opening may become narrowed to a mere button-hole slit, the typical anatomic basis for a mitral stenosis. Deformity of the aortic cusps results most often in insufficiency of this orifice, but not infrequently it is narrowed as well. Careful inspection will reveal in most instances, small, recent verrucæ scattered on the surface of the distorted valves. It is generally assumed that the valvular lesions of advanced rheumatic heart disease are the result of repeated attacks of rheumatic infection, each episode being accompanied by the growth of new verrucæ on the valve leaflets already scarred from the previous infections. Thalheimer has suggested that, in addition to the factor of repeated infection, the alteration of the blood-flow about a diseased and inelastic valve, together with possible microscopic endothelial tears following unusual effort, may lead to the formation of minute verrucous thrombi, which subsequently become fibrosed. This process repeated and extending over many years may result in a serious valve defect. He explains in this way the appreciable number of cases of chronic cardiovalvular disease in adults who have a history of only a single attack of rheumatic fever in early life, or who give no history of antecedent infection. On the other hand the fact that the rheumatic infections are often so insidious and mild in their

outward manifestations suggests that those patients may have had repeated infections which have been overlooked

The relative frequency with which the different valves are affected is illustrated by the following figures, which are tabulated from a study of 62 autopsies at Montefiore Hospital on patients who died of rheumatic heart disease

Valve diseases	Number of cases
Mitral alone	21
Aortic alone	3
Mitral and aortic	19
Mitral and tricuspid	8
Mitral, aortic, and tricuspid	11
	—
	62

In this same series aortic stenosis occurred fifteen times, mitral stenosis forty-seven times, and tricuspid stenosis six times. The frequency with which the tricuspid valve is implicated is worthy of note. Of course this distribution of the valvular lesions is representative of the long-standing cases, not of the early ones.

With the development of valvular lesions dilatation and hypertrophy of the chambers of the heart appear. Probably in no type of heart disease, with the exception of adhesive pericardio-mediastinitis, do we see such large hearts. In the far-advanced cases all chambers share in the enlargement. In the presence of mitral stenosis, even of moderate degree, and auricular fibrillation, the auricles may be enormously distended. Not infrequently this is accompanied by thrombus formation in the auricles on the left side, in particular. Small thrombi occupy by preference the auricular appendage. In exceptional cases they may become so large as to occupy almost the entire auricle, leaving but a narrow passage between the afferent veins and the auricular-ventricular opening. The scars in the myocardium do not parallel the severity of the clinical symptoms of myocardial insufficiency.

Pericarditis is always accompanied by some degree of myocarditis and frequently associated with rheumatic endocarditis. It is a common effect of the localization of the rheumatic virus in the heart. Its incidence as determined at postmortem at the

Bristol General Hospital was, in patients dying in the first decade, 100 per cent, in the second, 83 per cent, in the third, 41 per cent, in the fourth, 23 per cent, after the age of forty, 26 per cent, all ages together, 53 per cent. It may be fibrinous, the whole heart and pericardium being covered with a plastic exudate of fibrin. More often it is accompanied by a serous effusion into the pericardial sac which may amount to over a liter. The exudate is inflammatory in character, with a specific gravity above 1.015, rich in albumin, and showing polymorphonuclear and mononuclear leukocytes on microscopic study. If the patient recovers, the fluid is absorbed and the fibrin becomes organized, leaving white patches of scar tissue on the epicardium. Very often adhesion between the two layers of the pericardium takes place, sometimes in localized areas, sometimes involving the whole surface of the heart. Organization of these adhesions occurs, resulting in fibrous union between the two layers of pericardium in whole or in part. This is a common pathologic finding in patients with long-standing rheumatic heart disease. More rarely the process extends beyond the pericardium into the mediastinum, and radiating bands of adhesions connecting the pericardium with the surrounding structures are formed.

Symptomatology —Stage of Active Infection —It is a matter of gravest importance and, at the same time, of utmost difficulty to determine during a febrile attack of rheumatic origin whether or not an active infection of the heart has taken place. If the lesion is overlooked, overexertion on the part of the patient may aggravate the malady, if heart disease is incorrectly diagnosed, the patient's activities may be unnecessarily curtailed and he may become a cardiac neurasthenic and never rid himself of his delusion. In every case, therefore, presenting evidence, be it ever so remote, of infection with the rheumatic virus, the physician must study the heart with particular care to determine whether or not it has become involved in the general infection.

The chances for error are manifold. The rapid pulse may be due to fever, a systolic murmur alone is not pathognomonic of endocarditis. Daily examination of the heart, however, may reveal significant changes in the physical signs. Very suggestive

is an increase in size of the heart accompanied by an apical systolic murmur. Cardiac dilatation, however, is not very common at this stage unless the infection is very severe. The first heart sound may become short and the pulmonic second sound accentuated. A rapid pulse out of proportion to the height of the temperature or, still more significant, a pulse-rate that persists over 100 after the disappearance of the fever, may be the only evidence of cardiac involvement. If the pericardium is affected, a friction-rub may be audible and give telling evidence of the cardiac infection. In other cases the discovery of a pericardial effusion may first draw attention to the heart. In children the severest and most persistent infections are associated with a serofibrinous pericarditis. This localization should be suspected in every case in which the course of the rheumatic attack is prolonged and cardiac failure progressive.

The most significant evidence of myocardial invasion is the appearance of an irregularity in the pulse. This is not always easy to detect, but if carefully sought for, particularly with the employment of instrumental aids, such as the electrocardiograph or polygraph, will be found in about one-third of the cases. Frequently the arrhythmia is caused by premature beats which instrumental records show are often auricular in origin. There may be a significant relationship between their frequency and the common development of auricular fibrillation in the advanced stages of rheumatic heart disease. Partial heart-block can usually be recognized only in the graphic tracing by noting the prolonged auriculoventricular (A-V or P-R) interval which normally does not exceed 0.2 second. Occasionally a dropped beat may be noted on auscultation, giving evidence of the complete blocking of an auricular impulse. Arborization block and bundle branch block are uncommon in acute rheumatic heart disease. The cardiac irregularities which have been described characteristically disappear even from the graphic heart records within a few weeks of their onset.

In addition to the signs enumerated above, which point directly to cardiac involvement during rheumatic fever, the patient usually presents signs of the general infection, such as

fever, prostration, and leukocytosis. A rapidly appearing secondary anemia is seen with particular frequency in the rheumatic infections.

In a case in which the evidence of rheumatic disease is slight, in which, for instance, there are joint pains without signs of local inflammation, or, as was pointed out by Bouillaud, in a case in which the local manifestations have regressed to normal, yet the signs of general infection which have just been described persist, cardiac infection is the most probable cause for the general intoxication.

Some of these patients may exhibit dyspnea and at times slight cyanosis. The anemia, however, is the most striking feature.

The problem of ascertaining the presence or absence of active cardiac infection during an acute febrile episode becomes still more difficult in patients whose hearts have already sustained organic damage in the course of a previous rheumatic infection. In such cases it may be impossible to determine whether the local signs of cardiac involvement date from the earlier infection or whether they are recent in origin. The diagnosis must rest on a significant change in the physical signs taking place under the eyes of the observer, or else on the febrile course in the absence of arthritis or other local manifestations which might account for the fever.

In all cases of obscure fever in which there is reason to suspect the existence of rheumatic carditis, a blood-culture is of great importance. No bacteria are recoverable from the blood of patients with rheumatic heart infection. The presence of a bacteremia points to the existence of a subacute infective endocarditis or to some other form of sepsis. Rheumatic endocarditis may further be distinguished from the infective form by the absence of the typical petechiæ and other embolic phenomena, and by the absence of splenic enlargement.

After the defervescence of the acute rheumatic attack the inflammatory process in the heart slowly becomes quiescent and the active lesions are replaced with scar tissue. The time that elapses before the infection of the endocardium, myocardium, or

pericardium becomes arrested and healed varies in individual cases. In most instances it takes a month. There are many cases, however, particularly in children, in which, as has been pointed out before, the rheumatic virus remains active for many weeks, one rheumatic infection following another in close procession. In such cases the heart, too, may be subjected to repeated invasions of the rheumatic virus, new lesions developing before the old ones have healed.

The end-result, so far as it concerns the heart, of any particular attack of rheumatic fever may be estimated two or three months after the attack. At this time complete healing of the inflammatory lesions will have taken place, and the damage to the heart will be represented by scarring of the myocardium of the valves and pericardium. It is at this stage of the process that the mechanical factors of valvular efficiency play a predominating rôle. The heart muscle itself rarely suffers serious anatomic injury from one attack of rheumatic fever. Its function is not impaired by the small scars which are the aftermath of the acute myocarditis. A fibroid incompetent valve, however, is a permanent mechanical impediment to the efficiency of the cardiac pump and entails certain secondary adaptations of the heart to the new conditions under which it must work. The degree of impairment of the circulation will depend on the severity of the valvular defect and on the functional sufficiency of the myocardium. A slight mitral or aortic insufficiency may be borne without any evident disability. Such a patient may go through life with slight or no cardiac enlargement, and with no symptoms of a weakened circulation. It is probable that in cases with minimal lesions there is a complete restoration to normal. Unfortunately, both of these results are exceptional. Ordinarily the initial mechanical defect is of sufficient degree to place a permanent handicap on the efficiency of the cardiac pump. Of much greater importance is the disposition of these patients to reinfection with the rheumatic virus. It is in these individuals that we see recurrence after recurrence of frank manifestations of the rheumatic cycle, with repeated injury to the heart. Of equal significance are the atypical and insidious recrudescences of rheumatic

infection, vague joint and muscle pains, mild sore throats, bouts of undetermined fever, which so often are accompanied by renewed infection of the heart

The later stages of rheumatic heart disease, the patients with mitral stenosis or multiple valvular defects, with auricular fibrillation and evidences of myocardial insufficiency, are the result of infections spread over many years, acting on a heart working under progressively greater mechanical disadvantages. Unfortunately, it is these late effects, primarily, which come under the physician's care. In the earlier stages, when the cardiac lesion is still minimal, the patient experiences no disability and does not seek medical advice

CHRONIC RHEUMATIC HEART DISEASE

In the preceding sections we have studied the period of active invasion of the heart by the rheumatic virus. There remains for consideration the residual lesion and the ultimate outcome of the infective process. Chronic rheumatic cardiac disease exhibits a multiplicity of clinical and pathologic pictures, valvular defects, myocardial or pericardial disease, alone or associated one with the other, may all arise from the common infection. The symptomatology and pathologic physiology of these lesions resemble in many respects those produced by similar anatomic lesions of different origin. At this point we shall emphasize those features which serve to differentiate the chronic cardiac disorders of rheumatic origin from those induced by other agents of disease.

Mitral Insufficiency.—Of all of the valvular lesions, this is the most frequently encountered. To establish the diagnosis there must be, in addition to the murmur, a clear history of rheumatism and evidence of cardiac enlargement, or a mitral configuration of the heart in the roentgenogram. Mitral insufficiency is usually the earliest sign of permanent damage to the heart by the rheumatic virus. It may be apparent a few weeks after the infective period. It is of little clinical significance if unaccompanied by other cardiac lesions. The heart soon accommodates itself to the new dynamic conditions, and for a

time, at any rate, maintains the circulation with little impairment of its reserve power. Patients with simple healed rheumatic mitral insufficiency may carry on for years without any symptoms of cardiac insufficiency. Unfortunately, because of the great tendency to reinfections of the heart, the lesion rarely remains stationary and, in the course of time, mitral stenosis or multivalvular lesions develop. It is well to remember that uncomplicated rheumatic mitral insufficiency rarely causes much cardiac hypertrophy or symptoms of cardiac weakness. If such be present, the damage to the heart is either more extensive from more advanced valvular lesions, myocardial weakness, pericardial adhesions or disorders in rhythm, or else the mitral defect is not rheumatic in origin. It may be a relative insufficiency accompanying hypertension or myocardial degeneration, it may be due to arteriosclerosis of the valve, or it may be caused by an active bacterial endocarditis.

Mitral stenosis, with rare exceptions, is rheumatic in its origin. An insufficiency of the valve almost invariably accompanies the narrowing of the orifice. The lesion is much more common in women than in men. It is encountered most frequently in the first four decades of life, but cases occurring in individuals past their fiftieth year are not nearly so uncommon as is generally assumed. In these older individuals one must be careful to exclude arteriosclerosis of the mitral valve as a rare etiologic agent.

Mitral stenosis compared with mitral insufficiency is a late sequel of rheumatic valvulitis. It is self-evident that many months, in many cases one or two years, must elapse before scarring and shrinking of the mitral valve will be of sufficient degree to narrow the auriculoventricular orifice. Indeed, in most instances, mitral stenosis is evidence that the patient has experienced several attacks of rheumatic infection. The lesion is of more serious import than mitral insufficiency, it places the heart at a greater mechanical disadvantage. Yet, not infrequently, one encounters cases in young women in whom the defect is discovered accidentally and in whom no symptoms have drawn attention to the existence of cardiac impairment. Such

individuals, if the lesion is non-progressive, and if they are spared further infection, may survive many years. In most cases, however, mitral stenosis is a progressive disorder causing ever-increasing disability to its subject.

The general appearance of these patients is usually quite characteristic—cyanosis of the lips of varying intensity and a dusky flush of the cheeks. When the lesion dates from childhood these individuals are often small, thin, and weakly due to retarded development. Dyspnea and palpitation are common features. Strangely enough, the palpitation, which often is very annoying, disappears with the onset of auricular fibrillation. As the disease progresses, increasing stenosis, as well as insufficiency of the valve, becomes manifest and, in most cases, fibrillation of the auricles sets in. The frequent incidence of auricular fibrillation in these cases may be related to the common occurrence of auricular extrasystoles during the period of active invasion. Auricular flutter in some cases precedes the fibrillation. In many cases lesions of the aortic valve, and, in fewer instances, defects of the tricuspid valve are associated with mitral stenosis.

Symptoms of pulmonary and peripheral stasis, particularly congestion of the lungs, chronic passive congestion of the liver, and cardiac edema, sooner or later are added to the clinical picture. Extreme dilatation of the auricles supervenes in many cases. This is often accompanied by the formation of intra-auricular thrombi, which may release emboli. Pulmonary infarction, with or without hemoptyses, is a frequent symptom of mitral stenosis and often leads to a false diagnosis of pulmonary tuberculosis. These pulmonary emboli are not always derived from the right auricle, but may have origin in the systemic veins. Cerebral embolism, with ensuing hemiplegia, is a common complication of mitral stenosis. The spleen and kidneys are the other favorite localizations of emboli.

In other cases adhesive pericarditis or severe myocardial damage accompanies mitral stenosis. It is evident, therefore, that full blown rheumatic mitral stenosis presents a very complex and varied clinical picture. Characteristic is the steady downward progress of the patient and the multiplicity of the associated lesions.

Aortic Valvular Disease—As is the case with mitral insufficiency, so rheumatic aortic insufficiency may arise early in the course of a rheumatic infection. In certain few cases the valvular lesion may be slight and non-progressive. Such patients, if spared recurrent infections, may continue for many years without much cardiac enlargement and without symptoms referable to the heart.

Case III—During the World War a lieutenant in a machine gun company, who was considered one of the best and most active officers in his battalion, was referred to me for physical examination prior to promotion. Examination of his heart revealed definite enlargement with a distinct diastolic murmur at the left sternal border. His blood-pressure was 160/40. There were no other signs or symptoms of heart disease, no throbbing of the arteries, no dyspnea even on severe exertion, and no precordial pain. For months he had taken his company on daily exhausting drills without experiencing the slightest distress. He gave no history of antecedent infection or cardiac weakness. The Wassermann reaction was negative.

This is a typical instance of the so-called "latent" or "silent" type of aortic insufficiency. The patient at some time apparently had had a mild rheumatic infection of the aortic valve, with very slight involvement of the myocardium. His heart hypertrophied and, because of the good musculature and the absence of repeated infections, was able to maintain the circulation, even under the severest stress. Such patients may go on for many years without experiencing any disabilities, but they are always in danger of further damage to their heart through infection, and of a gradual loss of reserve from overexertion. That is why the man was discharged from the army, although, at the time, he was able to carry on his work better than the average soldier. Such cases are not so very unusual and can be detected only by a routine physical examination with careful attention to the physical signs. Osler mentions a case of a man with such a "silent" lesion who was observed for thirty-five years, during which period he exhibited no symptoms of heart disease. More

often the patient with rheumatic aortic insufficiency quickly becomes aware of his lesion

There are certain features which serve to distinguish aortic insufficiency of rheumatic origin. It is pre-eminently a disease of the young. In individuals below the age of thirty aortic lesions are almost invariably rheumatic sequelæ. To be sure, it may be found in older patients as well, and then it must be differentiated from the syphilitic and arteriosclerotic forms. A second characteristic is the fact that rheumatic aortic insufficiency is rarely an isolated phenomenon. In most cases it is associated with other valvular defects or with pericardial involvement. When stenosis of the aortic orifice is associated with insufficiency of the valve, the lesion is almost always rheumatic in origin, rarely arteriosclerotic, and never syphilitic. The size of the aorta, as determined by fluoroscopy, is frequently normal, even in advanced cases of aortic insufficiency, whereas in the syphilitic and arteriosclerotic forms aortic dilatation is the rule. Percussion over the manubrium may give deceptive results in these cases. I have seen a number of patients with a major leak of the aortic valve, with tremendous vessel pulsation, who, to percussion, showed apparent aortic widening, yet who, on Roentgen examination, were shown to have aortas of normal size.

Angina pectoris is unusual in patients with rheumatic lesions of the aortic valve. The following case is illustrative.

Case IV.—The patient is a girl nineteen years of age. When she was nine years old it was noticed that she was weakly and below par. A physical examination then revealed an organic heart lesion. Before this she had had measles, scarlet fever, diphtheria, and whooping-cough, but none of the rheumatic group of infections. The following year her tonsils were removed. In spite of this she had occasional attacks of fever, and joint and muscle pains in the following years. She was admitted to the hospital at the age of sixteen, complaining of pains in the shoulder, dyspnea, and attacks of precordial pain.

Physical examination on admission. A young girl, very dyspneic, with a distinct pallor about the mouth and eyes, flushed

cheeks, and throbbing neck vessels. The heart is greatly enlarged, with a heaving apex impulse and a systolic thrill over the aortic area. At the aortic area is heard a rough systolic murmur and a soft diastolic murmur. A pistol-shot sound is audible in the brachial and radial arteries. Ever since her admission to the hospital the patient has had frequent attacks of anginoid pains. She is very neurotic and the slightest emotion or excitement upsets her, causing marked tachycardia and precordial oppression. During the attack the systolic blood-pressure rises to between 250 and 300 mm Hg. The following is a description of a typical seizure.

The patient suddenly experienced intense pain over the precordium, which radiated to the left side posteriorly and to the right arm. The breathing was rapid and was accompanied by dilatation of the *alae nasi*, the face became markedly flushed, with slight circumoral pallor, her head was drawn back, and she assumed the position of *opisthotonos*, which seemed to give temporary relief. The pulsations of the heart and of the peripheral vessels were greatly exaggerated, in fact, the whole body and the extremities were shaken with each heart beat. The lungs were clear, blood-pressure 300/0. Nitroglycerin was given, which caused a drop in pressure to 194. A few minutes later, however, the attack was renewed, the blood-pressure rose, and the patient complained of a vise-like constriction in her chest.

These attacks come on at varying intervals and with varying degrees of intensity and have continued to the present. At no time have there been signs of myocardial insufficiency, such as edema, congestion of the lungs, or enlargement of the liver. Definite zones of hyperesthesia are quite constant over the upper sternum, in the region of the apex, and in the left paravertebral area from the third to the sixth dorsal spine. During the intervals between the attacks the blood-pressure is approximately 190/0. An *x-ray* examination shows no enlargement of the aorta. This is especially interesting in view of the long duration of the aortic lesion and in view of the widened area of manubrial dullness. The important features of this case are the anginal seiz-

ures, associated with a marked vasomotor disturbance. It is very difficult to determine the nature of these attacks, but in view of the marked pulsation of the aorta, as well as of the other vessels, it is possible that they are associated with involvement of the root of the aorta.

Tricuspid Valvular Disease—Organic lesions of the tricuspid valve are almost always of rheumatic origin. They practically never occur alone, but are associated with aortic and mitral valvular disease. Valvular deformity of sufficient extent to cause physical signs and symptoms is distinctly uncommon, although small verrucae are often found on the tricuspid valve at autopsy, and relative insufficiency of the valve from relaxation of the auriculoventricular ring is frequently encountered.

Pulmonic insufficiency from rheumatic valvulitis is very rare. In patients with mitral stenosis the diastolic murmur of relative pulmonic insufficiency, described by Steell, may be audible at the pulmonic area. Characteristic of this murmur is its inconstancy.

Myocardium—Most patients with long-standing rheumatic heart disease experience some permanent damage to the heart muscle. The lesions, which are not of great extent, consist of multiple minute scars which replace the Aschoff bodies of the acute infective stage. Often they are visible only microscopically. Such a pathologic lesion cannot be called a chronic myocarditis, it is rather the healed stage of an acute or subacute myocarditis. There is no correspondence between the efficiency of the heart and the extent of the anatomic lesions. Indeed, it is the rule to find on autopsy of these individuals insufficient evident causes for the myocardial insufficiency. This problem has been studied carefully by Aschoff and Tawara, who reach the conclusion that there is no relation between the gross pathologic changes in the heart muscle and the functional competency of the heart. More subtle dynamic and physiologic moments that escape detection must play the dominant rôle in determining the functional sufficiency of the heart. In rare cases the scars, by involving the conducting system of the heart, produce disturbances of function, such as heart-block and bundle branch

block In a recent case of rheumatic mitral stenosis in a girl of sixteen observed at Montefiore Hospital the electrocardiogram revealed right bundle branch block For over a year before death the patient's cardiac reserve was very poor This is probably the youngest case on record of this lesion

The most common manifestations of myocardial affection in patients with chronic rheumatic heart disease are, first, auricular fibrillation, and, less often, auricular flutter The anatomic basis for these disturbances of rhythm are unknown Fibrillation and flutter of the auricles are found most often associated with mitral lesions, especially mitral stenosis In a series of 130 cases of rheumatic mitral stenosis 50, or 38.5 per cent, showed auricular fibrillation and 2, auricular flutter Of 52 cases of rheumatic aortic insufficiency 11, or 21.1 per cent, exhibited auricular fibrillation and none auricular flutter

Early in the evolution of rheumatic heart disease auricular extrasystoles or paroxysms of auricular flutter or auricular fibrillation may give warning of the subsequent permanent alteration in rhythm Symptoms of myocardial insufficiency in these patients often date from the onset of auricular fibrillation Once fibrillation is well established it usually remains permanent and adds greatly to the mechanical embarrassment of the heart

As has been pointed out before, there is no close correlation between the character of the organic defect and the efficiency of the cardiac pump Two patients may present almost identical physical signs, yet one will have a fair cardiac reserve, the other will be continuously bedridden Morphologic studies of the myocardium in appropriate cases offer no explanation of this phenomenon This is worthy of emphasis, for it teaches that the maintenance of an adequate circulation is determined not by the anatomic consequences of the rheumatic infection, but by unknown physiologic moments which at present escape detection

Pericardium—Adhesions between the visceral and parietal layers of the pericardium is of frequent occurrence in patients with chronic rheumatic cardiac disease Of 130 cases of rheumatic mitral stenosis, 8, or 6 per cent, had, in addition, adherent pericardium In only 2 of these cases, however, did this com-

plication give rise to symptoms. A simple coarctation of the two pericardial layers causes no embarrassment to the heart's action and can be diagnosed only at necropsy. When, however, the adhesions extend beyond this and fix the heart to the diaphragm or to the surrounding mediastinal structures they interfere with the normal contraction and emptying of the heart and call forth cardiac hypertrophy, which soon is followed by signs of a failing circulation, particularly if valvular lesions exist simultaneously. The physical signs and diagnostic criteria of adhesive pericarditis need not be discussed here.

At times a chronic pleuritis with or without effusion is associated with such pericardial involvement. Such pleural exudates may be differentiated from effusions due to stasis by the fact that they are persistent, that they do not clear up as the cardiac function improves, that they show usually a specific gravity over 1.015 and an increased albumin content.

Progress and Prognosis of Rheumatic Carditis—In rare cases the first rheumatic infection of the heart proves fatal. In some the primary infection leaves the heart with a partly crippled valvular apparatus, the heart becomes adjusted to the new dynamic conditions, the patient escapes further infection, and the lesions remain stationary. Such individuals may survive to old age without any disability. These eventualities are, however, distinct exceptions.

It is the rule that the first attack of rheumatic carditis forges the first link in a chain of events that results in a progressively increasing disability of the heart that sooner or later leads to the death of the patient. The most important factor in the evolution of rheumatic carditis is recurrent rheumatic infection. This may manifest itself in a frank attack of acute articular rheumatism, chorea, or endocarditis, or in a more insidious fashion. There may be many recurrences, each of which adds a little more to the existing lesion, leading to the final picture of a heart with extensive multivalvular lesions, possibly with adhesive pericarditis, with dilated chambers and hypertrophied and insufficient musculature. It is not the rheumatic infections alone that are to be feared, although they are the greatest source of

danger, particularly in children. Other intercurrent infections, such as bronchopneumonia or follicular tonsillitis, may damage the heart severely. They do not give rise to characteristic or recognizable lesions, but they serve greatly to impair the cardiac reserve, and often determine a temporary or even a terminal breakdown.

Another common and important cause of the downward progress of these patients is auricular fibrillation. Itself often a result of rheumatic heart disease, it adds tremendously to the handicaps under which the diseased heart is laboring. The very rapid heart action, which, because of its gross irregularity greatly reduces the efficiency of the cardiac pump, throws an added burden of unnecessary work on the weakened heart, which soon becomes unable to maintain the circulation.

Finally, excessive bodily effort, by overtaking the heart already diseased may further impair its efficiency. It is doubtful whether any but the most extreme overexertion can injure the normal heart. But the diseased organ has a more limited range of response to the demands placed on it by active physical exercise, and is easily overtaken. Thus an attack of myocardial insufficiency is often conditioned by physical overwork. In women pregnancy, by overloading the heart, often determines cardiac failure.

The progress of every case of chronic rheumatic heart disease is therefore determined largely by three factors: reinfection, the onset of a gross irregularity, or excessive bodily effort. The evolution of the visible valvular or myocardial lesions depends on the frequency, intensity, and chronicity of the reinfections. The ability of the heart to maintain the circulation is conditioned just as much, however, by the other two factors. The unfavorable influence of auricular fibrillation is readily recognized and easily controlled in most cases by the administration of digitalis. The deleterious effects of overwork, which are more intangible and cannot be accurately measured, are none the less real. To a certain extent these several factors run parallel. The more advanced the organic lesion, the greater is the probability of the onset of auricular fibrillation, the greater the likelihood that even minimal effort will act unfavorably on the heart.

It is evident from the above considerations that the downward progress of most cases of chronic rheumatic heart disease is slow and intermittent. Gerhardt studied 123 cases with a view of determining the time which elapsed between the onset of the heart disease and the first symptoms of cardiac insufficiency. The following table gives his results

Duration of compensation	Number of cases
0- 5 years	37
6-10 "	33
11-20 "	30
21-30 "	9
31-40 "	12
41-44 "	2
	<hr/>
12 years' average of	123

After recovery from the primary infection the patient is usually up and about in good health for a variable period, which may last months or years. He then experiences a renewed attack of rheumatic fever which leaves the heart still more damaged. It is usually at this stage that symptoms such as dyspnea and palpitation appear, giving evidence that the heart no longer has its normal range of response. If now the patient constantly overtaxes his strength, symptoms and signs of myocardial insufficiency will arise. With appropriate treatment the heart's function may be restored, but renewed insults sooner or later will determine another breakdown. Each time it becomes more difficult to regain the ground that has been lost, and eventually the organic and functional disturbances of the heart become so great and its ability to meet the increased circulation needs induced by even slight exertion so limited, that the patient remains confined to his chair or bed. In most patients many years elapse before this stage is reached, and much can be done to retard the downward progress of the disease.

Coombs has made a valuable contribution to our knowledge of prognosis in rheumatic heart disease. He was able to follow up 600 cases of rheumatic heart disease for a period of many years, in not a few for as many as fifteen years. Of the children

who, when first seen, gave indubitable evidence of rheumatic carditis, 21 per cent died within ten years, while in a similar series, consisting of children in whom the signs of cardiac infection were described at the time of the first examination as doubtful and suspicious, only 6.5 per cent had succumbed in a similar period. On the other hand, one-third of the patients in both series developed unmistakable valvular fibrosis in the course of ten to fifteen years after the onset. It seems that the members of the first group were subject to more overwhelming infections than those in the second group, but that the insidious development of valvular disease is as apt to follow a mild, hardly recognizable rheumatic infection, as a severe one. These observations emphasize the gravity of the very mild rheumatic manifestations in childhood.

The age incidence of deaths, as calculated for a total series of 263 cases from Coombs' Table XIV, is represented in the following table.

Number of cases	AGE INCIDENCE OF DEATHS EXPRESSED IN PERCENTAGES						Average age at death years
	Up to 10	11-20	21-30	31-40	41-50	51 upward	
263	5.3	30.8	24.3	19	13.3	7.2	28.4

Contrary to the usual teaching, mitral stenosis is not at all uncommon after the age of fifty. Of 115 cases of rheumatic mitral stenosis at Montefiore Hospital, 32 were beyond this age. Auricular fibrillation always adds greatly to the gravity of the prognosis. Patients with aortic insufficiency in particular do badly once severe cardiac insufficiency or fibrillation has set in. Rheumatic heart disease is more serious when it develops early in life.

The economic condition of the patient often plays a determining rôle in the progress of the disease. If the patient is under constant medical supervision, if he can spare himself and so minimize the chances of reinfection or of overexertion, many years may be added to his life. If, when auricular fibrillation sets in, the ventricular rate is kept under constant check by the intelligent administration of digitalis, this irregularity loses much

of its baneful influence. It is apparent that a poor patient who cannot afford to spare himself but who continues to work until symptoms of heart failure appear, hastens the progress of the disease. This is the reason why women with valvular lesions live to a riper age than do men, the wage-earners. Of 88 men with rheumatic heart disease admitted to Montefiore Hospital, only 10 per cent. were over fifty years of age. During the same period 111 women were admitted, of whom 22.5 per cent. were over fifty years old. I have repeatedly seen patients with far advanced heart disease who, while they were retained in the hospital, continued up and about in a condition of comparative comfort and safety, but in whom, as a rule, within a month after their discharge, severe heart failure set in, which brought about an early death. Such individuals should be retained in a custodial institution for the rest of their lives. The temperament of the individual is another important element. As a rule, a person with a phlegmatic disposition will do better than one who is nervous and high strung.

- Various unforeseen accidents may hasten a fatal issue. Chief among these are emboli, which may lodge in the lungs, the brain, or in the arteries of the extremities. These occur almost exclusively in patients with mitral stenosis. In a sizable number of cases subacute *Streptococcus viridans* endocarditis becomes engrafted on the valve, deformed by the rheumatic infection and the secondary infection leads to the patient's death.

Rare cases have been described by careful observers who have followed the growth of a child to adult life, of apparent complete recovery from severe valvular heart disease, as far as could be ascertained by physical signs and symptoms.

Prophylaxis and Treatment.—*Prophylaxis*—In recent years much attention has been directed to the prevention of rheumatic heart disease. Societies for the prevention of heart disease have been organized and much money and effort have been expended in this direction. The movement has been of value in focussing the public mind on the importance of the problem, but little progress can be made until the questions of the etiology of rheu-

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matic fever and of its mode of entrance into the body have been solved

Most of the efforts of those who are endeavoring to prevent the development and progress of rheumatic heart disease are directed to the removal of foci of chronic infection which are supposed to be responsible for the recurrent infections. The tonsils, in particular, are regarded as portals of entry for the rheumatic virus as well as breeding places for repeated infections. The frequent association of tonsillitis with the onset of rheumatic infection does suggest a close relationship. Evidence that the so-called "chronic infected tonsil" plays an important rôle in the *recidives* is not so convincing. Crowe and his co-workers, in a careful study of patients whose tonsils had been removed, reported that of 25 patients who had had rheumatic fever, recurrences took place in 4 after tonsillectomy, and that of 24 patients who had had chorea, the symptoms returned in 11. These recurrences occurred in from three weeks to three years after the operation. On the average, the patients were under observation only two years.

More recently St Lawrence has reported a more intensive study of 85 children who were observed during an average period of three and one-half years after the operation was performed. Thirty-one of these children, or 36.5 per cent, showed recurrences in this period. Recurrences were more frequent in those with chorea than with the other rheumatic manifestations. St Lawrence stresses the fact that many patients who had had constant and frequently recurring manifestations for a period of years before the operation remained free of symptoms thereafter. On the other hand, he reports cases in which there had been no history of tonsillitis or any rheumatic manifestations before the tonsillectomy was performed, and yet which, years after complete tonsillectomy, showed acute rheumatic fever or some other allied manifestation. Many patients develop acute pharyngitis instead of tonsillitis after their tonsils have been removed. The indications for tonsillectomy have been defined as follows: (1) Hypertrophy, (2) evidence of infection, (3) enlargement of the tonsillar lymph-nodes, regardless of the size or ap-

pearance of the tonsil, (4) when the tonsils are the site of recurrent inflammation, regardless of their size or appearance and regardless of the size of the tonsillar nodes

A strict adherence to these "indications" would condemn almost every child to a tonsillectomy. Indeed, many misguided enthusiasts tonsillectomize, as a matter of routine, every patient with heart disease. In my experience most tonsillectomies, whether on sufferers from heart disease or on others, are unwarranted. The evidence that removal of the tonsils will prevent recurrences of rheumatic infection is not convincing, and the operation is not so innocuous as many would have us suppose. Many children require months to recover from the general deleterious effects of a tonsillectomy. Indeed, Hunt and Osman, who have studied the after-histories of 144 patients who had been patients in Guy's Hospital, with acute rheumatic fever, found a greater percentage of relapses of manifest rheumatic fever in the patients whose tonsils had been enucleated. Of 166 patients whose tonsils had been completely removed, 35, or 53 per cent, had definite recurrences, while of 78 non-operated patients, 33, or 42 per cent, had renewed rheumatic attacks. Their figures seem to show that recurrences are more frequent in patients with rheumatic heart lesions. This suggests the possibility that the virus may remain latent in the heart and from there initiate a new systemic infection.

In my experience the major indications for tonsillectomy in patients with rheumatic heart disease are the same as for those with normal hearts. They are repeated attacks of tonsillitis, badly infected tonsils with definitely enlarged tonsillar lymph-nodes, and tonsils which mechanically obstruct respiration or deglutition. In case of doubt it is wiser to postpone operation.

The teeth have been talked of a good deal as foci of infection in these cases. It is questionable whether infected teeth are related to rheumatic heart disease. However, every patient, whether he be a cardiac or not, should have his teeth attended to. Except in so far as this will improve the general condition of the patient, no striking effect on the cardiac lesion is to be looked for.

In recent years many cardiac convalescent homes have been established, particularly for children with rheumatic heart disease. It has been hoped that sending such children to these homes for a period of time would build up their cardiac reserve, and improve their general nutrition and resistance to such an extent that they would be less liable to renewed rheumatic infection. In children reinfection is by far the most important factor that determines the progress of rheumatic heart disease. While it is true that children who have had the advantage of a period of residence in these convalescent homes, as a rule, do remarkably well while they are inmates of the institution, in my experience they are just as subject to reinfection on their return home as they were before. Any child from a poor home will benefit by a rest in the country, coupled with good food and a well-regulated life—and in so far as they achieve this the cardiac convalescent homes accomplish a useful purpose. Whether they exert a permanent staying effect on the progress of the heart lesion in their little patients is open to serious question. If a rheumatic child of the tenements could permanently be given the advantages of a life offered by one of these convalescent homes, the outlook would be more hopeful. Something along these lines has been suggested by Raven, who would give these patients prolonged convalescent care for one or two years, analagous to that given in tuberculosis sanatoria.

At present convalescent homes accept, in particular, undernourished ambulant patients. They would be performing a more useful function if they accepted hospital patients who were just recovering from a rheumatic carditis. The general hospitals are unable to retain these patients for a long enough time, and often discharge them to their homes before complete healing has taken place. If such patients could be given prolonged careful convalescent care not only would healing take place with less permanent damage to the heart, but the frequent lighting up of an incompletely healed lesion would be less common.

There are no specific methods of therapy for rheumatic heart disease either in the acute or chronic stage. The salicylates, which are of such striking benefit to patients with acute articular rheu-

matism, have no effect on the heart, either in forefending infection or in arresting its course. The treatment must be symptomatic, with the methods ordinarily employed in cardiac insufficiency. Of these, rest is the most important.

CLINIC OF DR DANA W ATCHLEY

PRESBYTERIAN HOSPITAL

THE TREATMENT OF CHRONIC NEPHRITIS

IN proportion as disease is thoroughly understood is it properly treated. We know the etiology of diphtheria and we can produce an antitoxin that controls it. Our entire contact with such a case can be on a plane of rationalization, a plane of accurate knowledge of the cause and its mechanism of action. This seems to be very near perfection in therapy, but, unfortunately, there are very few conditions so well comprehended. Other methods of attack to meet defects in knowledge have long ago been developed. Some of them are based on unexplained experience, "it has worked, therefore it will work." This is pure empiricism and plays an important rôle in medicine. Science will eventually clarify these mysterious cures, as it did that of cinchona bark in malaria. We have spent years studying the effect of salicylates on rheumatic fever with no success, yet the value of this drug remains unquestioned. In some instances, however, we may very satisfactorily treat a disease, in spite of ignorance as to its cause, because we are familiar with its mechanism. The origin of diabetes mellitus is as obscure as ever, but the lack of insulin, which constitutes the troublesome effect of this disease, may be remedied. Cretinism is a similar affection. While, theoretically, we do not really stop such diseases, the practical results are extremely gratifying and permit us to offer an excellent prognosis.

The therapeutic approach to chronic nephritis is of still another type. Chronic nephritis is one of many diseases in which the causative agent, sometimes known sometimes unknown, produces a disturbance of normal physiology never to be re-

paired Our problem, therefore, is to readjust the load in proportion to the damage suffered by the machine that must carry it on. If the heart, for example, by some mechanical defect of valve or muscle cannot carry a man up one flight of stairs without dangerously overtaking its reserve, that man's life must be reorganized so that he will not meet a flight of stairs in his daily activities.

There is for chronic nephritis no known cause. Although there have been many suggestions and many fertile leads, the fact remains that we do not know the etiologic agents. We cannot remedy the physiologic disturbances produced by this disease, but we do understand them sufficiently well partially to readjust the organism to meet them. Therefore our proper approach, here, must be through a knowledge of the individual abnormalities in function presented by each patient, and our treatment will be concerned with the adjustment of that patient's life to meet his changed physiologic equipment. At times, of course, one must employ empiricism and use certain methods merely because they sometimes are effective, but first let us try the rational therapy based on a solid foundation of scientific knowledge, and only if this be unsuccessful, turn to the variety of procedures that we have inherited from our predecessors.

If, then, the treatment of nephritis is the treatment of a particular nephritic according to his individual variations, how are we to determine those individual variations and analyze their relative values? There are two fundamental requirements: first, an understanding of renal physiology with the clinical evidence of its disturbances, and, second, some familiarity with the course of the simplest types of nephritis.

For clinical use it is necessary to discard clinically insignificant classifications on the basis of pathologic anatomy. As a matter of fact, there has not yet been a completely satisfactory correlation of anatomy and physiology in the normal kidney, and attempts to prove a consistent relationship between the clinical course of chronic nephritis and the pathologic picture in the kidneys have been notably unsuccessful.

If we were hastily to survey the functions of the kidneys as

clinical studies we would begin with that of maintaining a normal amount of water in the body. Edema in Bright's disease is a disturbance of the water balance in which the kidneys may not be of primary importance, but nevertheless the kidneys certainly do not excrete water in a normal fashion. Restriction of fluids would therefore seem logical when this function is disturbed. Closely related to this matter of the water balance is a second function of the kidney, namely, the excretion of sodium chloride. For many years it has been known that there is some relationship between the retention of water and salt, whether the salt is retained to bring the edematous fluid to a normal osmotic pressure or whether the water is retained to dilute an excess of salt, is still disputed, but we do know that limitation of salt intake in edema is indicated by the disturbed physiology and is really helpful. May I digress to state that the so-called laws of chlorid excretion including the chlorid threshold, have no reliable experimental basis and it is a like fallacy to attribute any clinical significance to the plasma chlorid concentration. Indeed, modern work tends to show that the chlorine of the sodium chlorid is perhaps not the pathologically significant portion of the molecule, but that the edema is related to the retention of sodium.

A third function of the kidneys is to assist in maintaining the normal hydrogen-ion concentration of body fluids by the excretion of acid and the formation of urinary ammonia. If an animal is fed hydrochloric acid a very large portion is excreted as ammonium chlorid formed by the renal production of ammonia under the stimulation of an increased acid load. It is clear that severe destruction of the kidneys will interfere to such a serious degree with the acid-base mechanism that clinical acidosis may result. Knowing the mechanism of the acidosis we can direct treatment along intelligent lines.

The ability of the kidneys to excrete a urine highly concentrated in waste materials is a function that becomes very significant when the progress of Bright's disease markedly impairs it. One liter of urine of a specific gravity of 1030 contains all the waste substances found in three liters with a specific gravity of 1010. It seems obvious that a polyuria must develop when the

classical nitrogen retention syndrome Here is a patient (to describe a typical case) in his early forties, who has had occasional morning headache, nocturia, and moderate dyspnea on exertion for two years, has lost weight gradually, and comes to the hospital because of sudden dimness of vision, giving up work then for the first time He was rejected for insurance fifteen years ago due to albuminuria Physical examination shows loss of weight, anemia, hypertension, cardiac enlargement, and thickening of peripheral arteries without calcification His eye-grounds show a blurring of the optic disks and retinal arteriosclerosis with one or two hemorrhages His urine has a constant specific gravity of 1010 to 1012, a faint trace of albumin, and one or two hyaline casts He has 3,000,000 red blood-cells and 40 per cent. hemoglobin His blood urea is elevated, his blood CO_2 is decreased, and his phthalein is 10 per cent His appetite is poor and he feels sick Upon analysis we discover two distinct pathologic processes first, a constitutional reaction to some unknown toxic process, as evidenced by headaches, poor appetite, loss of weight, choked disks, perhaps, also, the hypertension, and, second, an impairment of renal function, including concentrating ability and excretion of nitrogen, phthalein, and acids The constitutional reaction is not due to the renal changes Observation, in a case with polycystic kidneys, of the complete well-being over many years in the presence of severely damaged kidneys, is striking evidence on this point Furthermore, the frequent lack of parallelism between nitrogen retention and uremic manifestations is similar proof We must treat this advanced nephritic with the consciousness that his illness is a fatal one and his exitus imminent Between the patient with edema, suffering a very slight deviation from normal, and this very seriously ill person on the other end of the scale is a wide space filled with all possible combinations of the characteristics of both—any constitutional symptom or all may be lacking, and any degree of renal impairment may be found Our treatment depends on the number and type of the various factors combined in any given case

It should be clear from the discussion so far that the treat-

ment of nephritis requires, first, an analysis of the picture presented by the case at hand, and, then, an application of certain general principles suggested by this analysis

It is needless to dwell upon such routine procedures as the elimination of foci of infection, restriction of activity and rest in bed, and we shall, therefore, turn directly to the matter of diet. Any nephritic suffers from enough involvement of the kidneys to justify saving them from unnecessary work, so, in every instance, lighten the load by a moderate restriction of protein and salt. This means, in practical terms, one helping of meat once a day, only one egg a day, very little cheese, and removal of the salt shaker. Further regulation of the diet is guided by a knowledge of the nature and degree of the disturbance of renal function.

Edema demands a restriction of fluid and salt. The fluids should be about 1200 c c a day. No salt should be added when cooking, butter should be fresh, salty foods eliminated (do not forget that baker's bread contains a good deal of salt), and very little milk allowed, none to drink. As a matter of fact, there is little place for milk in the whole field of chronic nephritic therapy. Milk contains too few calories, too much protein, and too much salt. It is rarely rational to employ in chronic Bright's disease the Karrell diet so useful in treating acute cardiac decompensation.

Loss of concentrating ability should be combated by giving the body more water for the kidneys to use. Fluids should rarely drop below 1800 c c. a day when this disturbance is present, a rule that takes precedence over the restriction of fluids, even though edema be present. For edema can do little harm in itself, but the kidneys must have enough water with which to excrete the waste substances constantly being formed.

Nitrogen retention calls for protein restriction, but the problem has not been solved when all meat and eggs and cheese are eliminated or greatly restricted. Our objective is to decrease the formation of nitrogenous waste substances in the body, and these substances come from two sources: protein food and the metabolism of tissue proteins. Both sources must be controlled.

As protein substances are forbidden, sufficient foods of other kinds must be substituted to bring the total caloric intake high enough to prevent the use of body protein for ordinary daily fuel needs. With the usual poor appetite this often becomes a difficult problem. In that event, cream, jellies and preserves are useful, and fruit juice drinks with fairly large quantities of lactose in them are easily taken. One must be very cautious, however, not to exceed gastric tolerance in a degree that produces vomiting, for, once induced, vomiting is very difficult to control. But because it is a mistake so often made, the injunction should be repeated. Do not starve a nephritic at any stage of his disease.

Acidosis usually occurs when the prognosis is so grave that treatment is futile. Rare cases have been prolonged for a time by the administration of sodium bicarbonate, with care not to upset a sensitive stomach.

If edema is a stubborn symptom, it is customary to consider the use of diuretics. The action of such drugs as theobromin and theophyllin depends on a stimulation in the kidneys of certain normal functions that are greatly suppressed in renal edema. It is not reasonable to expect uniform success in such an event, indeed, experiment and clinical observation have demonstrated that actual harm may be done to the kidneys by the unwise use of these diuretics, even though water excretion be temporarily increased. There is one type of diuretic that rests on a fairly sound theoretic and practical basis, viz., the use of calcium salts. Professor Blum, of Strassbourg, has suggested that edema is due to the retention of sodium, and his experiments seem to demonstrate that NaCl increases edema, KCl decreases it, NaHCO₃ increases it, and CaCl₂ decreases it. In other words, while salts of sodium retain water in the body, salts of potassium or calcium cause it to be excreted. As potassium is toxic, we use calcium chlorid or lactate, and extraordinary diuresis may be produced. These salts are often very disturbing to the stomach, so that not every case is available for their use. The lactate is the easier therapeutic agent and can be administered in doses of 5 to 10 gm a day. Ammonium chlorid (in the same dosage) may

be used when there is no nitrogen retention. When it works it is marvelous, but all cases do not respond. Like most diuretics, the action is not cumulative, if an immediate result is not obtained when the maximum dose is reached, discontinue its use.

One cannot discuss the treatment of nephritis and overlook the letting of blood. There is no therapeutic procedure that helps the anxious relative more than a spectacular phlebotomy. At times the temptation to the hard pressed physician is irresistible. There is one word of caution. Be sure the patient is not already anemic. Bleeding is of great aid in acute nephritis when a sudden shut down of renal function occurs, but it does little good in the chronic disease.

It is well to inquire into the physiology of sweating. If a greater concentration of waste substances can be excreted by the skin than by the diseased kidneys, then it would obviously be advisable to stimulate sweating, so that a portion of the water ordinarily eliminated by the kidneys might be diverted to this more effective channel. The facts, however, are all in favor of the kidney, even when severely damaged, it does better work than the skin. Moreover, sweating weakens the patient and exposes him to the danger of taking cold. One might add that uremic convulsions have occurred after sweating due to the concentration of toxic substances in the blood following great loss of water through the skin. Sweating is neither rational nor effective.

It is impossible to include any comprehensive observations on the treatment of complications of chronic nephritis. But there is a fairly frequent one of such importance that one cannot conclude without commenting on it. Cardiac decompensation is this complication, and it is significant because it may greatly influence both diagnosis and treatment. One can never be certain of the degree of renal impairment in the presence of passive congestion of the kidneys. Prognosis must, therefore, be guarded until further studies can be made with an improved circulation. Heart failure is a predominant indication for treatment—all other indications become secondary—the all-engrossing problem is that of restoring cardiac function. It matters little what other conditions are present.

No one studying or practising the treatment of chronic nephritis can fail to appreciate the helplessness of the physician in a large number of cases. Our results are often most discouraging. The numerous advances of the last twenty years have helped the patient mainly by eliminating many unpleasant therapeutic procedures and by making life more comfortable for a longer period of time. In contrast, however, to the meager benefit derived by the diseased individual, is the very great value of this progress to the physician. We are now able to understand the physiologic background of the clinical picture and upon such a rational basis determine the true value of our treatment. It is just this ability to interpret the relation of cause and effect that introduces the intellectual stimulus which saves any work from boredom. It is cheering to observe that, historically, this scientific step never far antedates the development of more practical phases of therapy.

CLINIC OF DR WALTER P ANDERTON

PRESBYTERIAN HOSPITAL

SYPHILIS OF THE CENTRAL VASCULAR SYSTEM

THIS afternoon we are to consider some very interesting manifestations of tertiary syphilis. Syphilis of the central vascular system has been known pathologically for many years, but it is only within the past fifteen years, due to the work of Allbutt, Blumer, Lamb, Longcope, and others, that the diagnosis and treatment has attained a rational clinical basis. Syphilis of the aorta and heart is a common occurrence, not difficult for you to recognize and treat. Let us first examine four patients with various manifestations and complications of this disease, and then proceed to some didactic remarks upon the subject.

DEMONSTRATION OF PATIENTS

Case I—This woman is forty-four years old, and is married for the second time. She was born in Germany. She denies primary or secondary lesions. Her only pregnancy terminated normally. She was married to her first husband for seventeen years. He died of heart disease, and she recalls that he was treated by injections into the muscles and veins a short time before he died. Her second husband is well. This well illustrates negative history of primary and secondary lesions. The syphilis was discovered by the routine Wassermann test when she came to our surgical clinic in 1921. At that time she received no antisyphilitic treatment because she left the city.

She returned again in 1923. At that time she complained of shooting pains and weakness of the lower extremities, and of poor eyesight, particularly at night. She had had some difficulty in walking for about a year, and was dyspneic when she

first arose in the morning. There was no nocturnal dyspnea. She coughed up considerable yellow sputum, and had palpitation after coughing. Argyll-Robertson pupils were evident.



Fig. 81—Six feet film of heart. Widened supracardiac shadow, suggestive of dilatation of the ascending portion of the aorta. There seems to be thickening of the interlobar pleura extending across the right lung field. Midline to right cardiac border, 5 cm. Midline to left cardiac border, 8.7 cm. Total transverse heart measurement, 13.7 cm. Width of great vessels, 7.2 cm. Internal diameter of chest at level of left diaphragm, 25 cm. (Dr. Ross Golden).

You will see that her pupils do not respond to this bright electric light, even when it is held very close to the eye. The knee-jerks are hyperactive, and equally so. The heart is regular and forcible. The apex-beat measures 10.5 cm. from the midline,

and dulness is 4.5 cm to the right, and 6 cm to the left in the second space. The left limit of cardiac dulness corresponded to the point of maximal impulse. Systolic murmur over the precordium, and loudest at the aortic area. The pulses are equally regular and forcible. Blood-pressure 130/75. Both kidneys are palpable, but the liver and spleen are not. Eye-grounds negative, and no Romberg's sign. The Wassermann has become negative after considerable treatment, and the urine is normal. An electrocardiogram failed to show any evidence of myocardial damage.

The x-ray shows a widened cardiac shadow (Fig. 81), dilatation of the ascending portion of the aorta, and thickening of interlobar pleura extending across the right lung field.

Spinal puncture was made in 1923, the globulin was negative, the Wassermann was negative in both antigens, and the Nonne test was 111,000,000,000.

Diagnosis—Syphilis of aorta and cerebrospinal meninges.

This patient complained very bitterly that mercury injections caused extreme discomfort locally, so we gave her bismuth. She has now had twenty-seven injections. We have had to be very careful in the administration, using long intervals and small doses, because she readily develops stomatitis and gingivitis. These are both symptoms of bismuth poisoning, as are diarrhea and renal upsets. All these must be carefully guarded against. This is also true with mercury. For this reason blood chemistry and phenolphthalein tests are helpful, and also urinalysis.

Case II—This man is a bill collector, aged sixty-two years, born in New Orleans, and of French descent. He is a widower. He first came under our observation July 30, 1924. At that time he complained of boring pain in his back, worse on walking, this he had noticed for about six years. His family history was quite irrelevant. His best weight had been 135 pounds, but at the time of his first examination here he weighed only 99 pounds, having lost weight gradually for about five years. His habits were good, he drank only two cups of coffee a day, took no alco-

hol, and ate and slept regularly. Bowel movements were also regular. His work naturally entailed considerable walking, both on the level and up and down stairs. He had had typhoid at



Fig 82—Stereoscopic films of the chest show rather prominent and beaded bronchial markings extending well into both apices and apparently reaching the pleura on the left, where they are the more conspicuous. The right diaphragm is a little irregular, looking as if it were held up in the inner third and its outer margin of adhesions. A prominent shadow in the right hilum region presumably represents enlarged glands. From these films alone one would be a little suspicious of an early bilateral apical, though it would be interesting to repeat this examination some time in the future (Dr E C McCoy)

thirteen. His gall-bladder and appendix were removed in 1914. When he was twenty-two he had had specific urethritis (gonorrhea), which was cured in about five weeks. There is no history

of chancre The Wassermann reaction in the spring of 1923 was 4+ At that time he received twelve mercurial injections in six weeks in Denver, after the diagnosis of aortitis had been made by Dr Ryan of that city



Fig 83—Normal cardiac outline Supracardiac shadow seems rather wide, but not quite what one would expect in aortitis, but rotation of patient on film is confusing Dilatation of aorta confirmed by fluoroscopy Midline to right cardiac border, 3.5 cm Midline to left cardiac border, 8.8 cm Total transverse heart measurement, 12.3 cm Width of great vessels 7.3 cm (Dr C. C McCoy)

Findings at Examination—The heart was regular and forcible, the heart dulness wide in the first and second spaces first space, 5 cm on the right, and 6 cm on the left, second space, 6 cm on the right, and 4 cm on the left The measurement was 10

cm from the midline in the fifth space The first sound at the apex was dull and distant, with visible pulsations over the second space

The pulsation over the left common carotid was considerably more marked than usual There was no tracheal tug or respiratory pulsations Diastolic shock was noted in the suprasternal region over the aorta

The lungs showed evidence of chronic pulmonary tuberculosis, moderately advanced A nodule was found in the left epididymis Hemorrhoids were present Knee-jerks were present under reinforcement

The x-rays show rather prominent and beaded bronchial markings, extending well into both apices, and apparently reaching the pleura on the left The right diaphragm is a little irregular, as if held by adhesions A pronounced shadow in the right hilum presumably represents enlarged glands From these films alone Dr Golden said that he would be suspicious of an early bilateral apical tuberculosis (Fig 82)

An x-ray of the heart (Fig 83) was not absolutely typical of aortitis The supracardiac shadow was slightly widened The ascending part of the arch of the aorta seemed to have a definite but no extensive localized dilatation, which probably indicates syphilitic aortitis

The blood Wassermann was 3+ with alcohol, and 3+ with cholesterol antigen

Diagnosis—A diagnosis was made of moderately advanced chronic pulmonary tuberculosis, fibroid phthisis, syphilitic aortitis, and hemorrhoids

Treatment—This patient has received thirty-nine injections of mercury and has been taking potassium iodid up to tolerance, 25 drops, three times a day On March 1st he had a very severe attack of paroxysmal dyspnea early in the morning He was brought to the hospital in a taxi, and stayed in bed about two days His recovery was satisfactory Since then he has taken 10 drops of tincture of digitalis three times a day until last week At that time the iodid was alternated with digitalis, every three days

Before dismissing him, I wish you to note the seborrheic dermatitis of the scalp and chest, gingivitis and several superficial lymph-nodes

Case III—This jaundiced patient emphasizes one of the other complications of this condition namely, syphilis of the liver. He entered the Presbyterian Hospital in October, 1920, at the age of fifty-three years. He had been married eighteen years. At the time of his admission he had pyorrhea alveolaris and irregular heart with occasional premature ventricular contractions. The cardiac dulness was wide at the base 6 cm on right, 5 cm on left in the first space, 4.5 cm on right, 6.3 cm on left in the second space. The left limit of cardiac dulness was 10.5 cm from the midline in the fifth space. The first sound was strong. The aortic second was louder than the pulmonary second. A soft localized systolic murmur was detected at the aortic area, louder when the patient leaned forward. In conducting an examination you should always have the patient bend over forward, with the trunk parallel to the floor, because this will intensify a murmur at the base, or let you hear an otherwise inaudible one. If the patient is in bed, have him turn over on his hands and knees. Slight visible pulsation was noted in the right second space, close to the sternum, no thrill, and no shock.

The pulses were equal and forcible, but irregular in rate and rhythm. A remarkably thickened wall was noted. Both brachial arteries were visible. The right kidney was the only palpable viscus. A left varicocele was found, and a serpiginous tertiary lesion on the left upper thigh, and posterior surface of the scrotum.

An x-ray of the heart showed the measurements to be as follows: from midsternal line to the extreme right border, 5 cm, to the extreme left border, 7.5 cm, the total diameter of the arch, 6 cm.

The electrocardiogram showed a rate of 88, P-R interval, 0.16, some left preponderance, and ventricular premature contractions of right or basal type, which tended to occur rhythmically every third beat. T-wave upright throughout.

The blood Wassermann was 4+ in both antigens. The patient had had a chancre twelve years before admission here. This was followed by an ulcer on the tongue. There had been no constitutional treatment for either primary or secondary syphilis.

The *diagnosis* was syphilis of the aorta, and general arteriosclerosis.

Treatment—This patient has received three full courses of mercury and arsphenamin. At the time he was first treated we were giving the old form of arsphenamin, not neo-arsphenamin. He received 5 gm. in all, and about 22½ gr. of mercury salicylate. It was necessary to stop the treatment at one time because of intense jaundice which developed shortly after one of the arsphenamin injections. This is at times a manifestation of arsphenamin intoxication.

Last September this patient became icteric. At first we thought this might be a late effect of the treatment, but after watching him carefully, we decided that the jaundice was due to syphilis of the liver. He had not had mercury for a year, with the exception of 2 compound cathartic pills, and he had had no arsphenamin for even a longer period. His liver was found to be enlarged and uneven, with large nodules which are common in tertiary syphilis of the liver.

Since he had become jaundiced during the earlier courses of arsphenamin, we now gave him bismuth rather than arsphenamin or mercury, to avoid the possible toxic action of the two latter drugs on the liver. With this treatment with bismuth, potassium tartrate, and butin he is feeling much better and is carrying on his work as carpenter. His vigor is increasing. The blood Wassermann is negative with alcohol, and 1+ with cholesterol antigen. The jaundice is not of an obstructive type, since his stools contain bile. Rhubarb and soda and other cathartics keep his bowels open, and he is drinking a great deal of water to wash out bile through the kidneys.

With the development of complications after treatment for four and a half years, the prognosis is not good.

Case IV—This patient, a Danish West Indian negro, has

syphilitic aortitis and general arteriosclerosis. He is forty years old and married. The arteriosclerosis is rather advanced for his age, probably owing to the syphilis.

He first came to the hospital in 1921, complaining of pain in the hips, arms, and weakness in the knees. These symptoms had existed for two years but had been worse during the last six months. His wife and 6 children were all well. No children had died and the wife had had no miscarriages.

The patient denied syphilis, but acknowledged gonorrhea, contracted twenty-five years previously. The Wassermann reaction was 4+. His blood-pressure was 120/80. Examination of the heart showed the first sound at the apex to be rough, and a slightly accentuated second aortic sound was noted.

A diagnosis of syphilis was made, and the patient was treated with mercury, iodid, and salvarsan. His symptoms were soon relieved, and he returned to work.

However, in August of the same year, he had to be admitted to the ward because of severe headaches which had persisted for fifteen days, and pain in his arms, forearms and shoulders. The pupils reacted to light, but the right one was slightly irregular in contour. He had poor teeth and a coated tongue. The cervical lymph-nodes were moderately enlarged. There was marked suprasternal pulsation, but no tracheal tug. The heart was enlarged to the left with a wide area of dulness in the first and second interspaces. There was a diffuse systolic impulse with a fine systolic thrill at the apex. The first sound at the apex was rough. There was a loud booming aortic second sound and blowing systolic murmur across the base. The blood-pressure was 240/98. The lungs showed a slightly impaired resonance at the right apex and diminished breathing at the right base. A small irregular punched-out scar was on the glans penis. The biceps, triceps, knee-jerks, etc., were normal.

Diagnosis—Syphilis, general arteriosclerosis, hypertension, cardiac hypertrophy and dilatation, aortitis, aneurysm of arch of aorta, syphilis of the central nervous system, probably meningal. Rapidity of progress of physical changes since first examination was very striking.

Treatment—He was treated with mercury and salvarsan. During the first stay in the ward this man had several attacks of severe pain over the precordium, with dyspnea. At one time there was also pain in the abdomen. The patient had marked icterus and an enlarged liver. Following the jaundice he became quite demented, at first very apprehensive with delusions of persecution, later his mental state changed to one of depression. During his stay in the hospital this man developed occlusion of the bifurcation of the aorta. It was impossible to feel the femoral pulse on either side, and compensatory circulation developed, some of which persists today.

We feel that this man was probably an example of so-called obliterating aortitis, of which a series has been reported from the Canal Zone by Darling and Clarke (1915). Patients with this rare manifestation of central vascular syphilis have gradual clot formation on the face of their ulcers, and obliteration of branches of the aorta. You can see now how these worm-like sclerotic superficial epigastric arteries stand out. At the time of the occlusion, his compensatory circulation showed as prettily as in any anatomic text-book. The superior epigastric branches of the internal mammaries ran out along the surface and pulsed visibly.

Examination of the heart showed systolic pulsation visible in the fifth and sixth interspaces, and in the epigastrium. P M I visible and palpable, heaving 10 cm. to the left in the fifth space. There was marked systolic thrill at the apex, and a very faint one at the base to the left, and just above the sternum.

The heart sounds were loud and forcible. The second sound at the base was snapping and valvular in quality, the aortic second was more accentuated than the pulmonic second. At the apex and along the left border of the sternum was a systolic murmur, which became louder and rougher when the patient sat up, and which was not transmitted to the axilla or back. The first sound inside the apex also had a rough quality when the patient sat up. A soft blowing systolic murmur was audible at the base, and transmitted upward over the great vessels. There was no diastolic murmur.

You will note the visible pulsation of the arterial vessels of this patient's neck, and the small pulsating vessels on the abdomen. In each lower quadrant, running upward from the middle of Poupart's ligament is a raised, tortuous pulsating vessel, the superficial epigastric. The radials and brachials are also visible. There are dilated, superficial veins on the arms, and across the upper anterior thorax. Slight tenderness and marked pulsation in the epigastrium.

After the patient left the ward, completely cured mentally, he worked at home and came to the hospital for treatment. Then he went back to the Danish West Indies, where he stayed two years. When he returned to the United States he was found to be very much improved. However, the last Wassermann, taken March 6th, was still 4+. It is felt to be advisable to continue treatment. We shall give him mercury injections, but probably no more arsphenamin. Bismuth will be used later. The patient is not taking any iodid at present, but he has shown that he can take 50 drops of saturated solution of iodid of potassium, three times a day, for several weeks without any intoxication.

LECTURE

Syphilis of the central vascular system deals principally with syphilitic aortitis, but this is often associated with myocarditis. The two conditions may occur separately or in combination. Men are affected three times more frequently than women; indeed, some authors give the sex ratio as 5 to 1. Laborious occupations predispose to central vascular syphilis, and the usual age incidence is from thirty-five to fifty years, although the disease may occur at any age. It may be congenital or acquired.

The exciting cause is the *Treponema pallidum* or *Spirocheta pallida*.

Pathology—Microscopically, perivascular round-cell infiltration is noted, commencing either in the adventitia or media, often between 2 and 4 cm. above the aortic cusps. One finds scattered areas of connective tissue with consequent loss of elasticity. All the coats are attacked.

In the gross, one sees the familiar yellow, round, and oblong

depressions, these are small, and have a definite margin which at times is elevated. Frequently there are coincident atheromatous patches. The disease may spread throughout the aorta, although it is most commonly found in the ascending portion. It may spread in either direction, extending to the coronary arteries, but seldom occurs below the diaphragm. The aorta becomes dilated as a result of loss of its elasticity, and also elongated. Sometimes this is spoken of as the Welch-Hodgson type of aortitis, also as fusiform aneurysm. Besides the dilatation there may be local puckering with formation of sacculated areas.

In addition to the aortitis, definite types of myocarditis occur as a result of tertiary syphilis. 1 Connective-tissue formation throughout the myocardium. 2 Perivascular infiltration, such as has been described in the aorta. 3 There may be milium gummata in the heart muscle.

Symptoms—Pain, palpitation, dyspnea, cough, asthma, cardiac insufficiency, fatigue, and loss of weight, with or without general malaise, are the usual symptoms.

The *pain* varies from a slight sensation of pressure, with embarrassment in the precordial region, to the other extreme of the most agonizing angina pectoris, with the fear of impending death. Patients who experience the latter type break out in a cold perspiration and, as they sometimes express it, feel as if an elephant had stepped on their chest. It is one of the most severe pains that man is heir to. This pain is felt not only at the primary site in the substernal or precordial region but also in the left shoulder and inner side of the left arm, or even in the forearm, little finger, and wrist. It may also extend over the left side of the chest and radiate up the side of the neck as high as the jaw and the temporomaxillary articulation. Sometimes an anginal attack is attended by salivation. Rarely the radiation may be to the right shoulder and arm.

Palpitation may or may not be brought on by physical or emotional excitement.

Two types of *dyspnea* are observed. 1 Exertional (precipitated by physical or emotional strain), 2 Paroxysmal-

nocturnal In the latter type, the patient wakes up in the night with a sense of inability to breathe He becomes orthopneic, and has an intense desire for more air This condition may be attended by cough, asthma, or even go on to pulmonary edema, but most attacks do not pass beyond the simple stage of dyspnea These paroxysms recur with increasing frequency, but may sometimes be controlled by proper treatment with digitalis

Coughing is an independent symptom which is frequently noted This is often attended by mucopurulent sputum

Cardiac insufficiency often develops in this disease It is of serious prognostic importance, and with it are found vertigo, dyspnea on exertion, orthopnea, edema, and anasarca These patients fatigue more easily than other people in the same pursuits, and tire earlier in the day than they were previously accustomed to Such patients are also considerably below the weight which they maintained a few years before

Signs—There are certain significant signs of syphilis of the central vascular system which vary in their combination In the first place these patients may or may not have elevation of temperature Some develop an unusual type of complexion with a peculiar sallow pallor, most likely to be found when aortic insufficiency is present.

Examination may reveal suprasternal pulsations, or an abnormally wide pulsation of the subclavian and common carotid arteries, or an aneurysm may cause localized pulsation There may or may not be a thrill over such an area of suprasternal pulsation, or over the aortic area, in the second right intercostal space The area of cardiac dulness is rather typical in its distribution It has been called "en casque," or helmet-shaped. The increased width of mediastinal dulness at the second space is due to the increased diameter of the aorta Sometimes a shock may accompany the pulsation

If the aorta is elongated, it is easy to understand that there is an abnormal mobility of the cardiac apex, and when the patient changes from the supine to the left lateral position the apex will move farther than normally This is an unusual sign

The heart may or may not be enlarged If there has been

long-standing hypertension, you will find an enlarged heart. However, in a simple uncomplicated case, it is common to find a heart of normal size. The aortic second sound is hollow. The French call it a *bruit de tabourka* (North African tom-tom). Sometimes there is a systolic murmur. When you find aortic valve insufficiency, the heart is enlarged downward and to the left. Then a diastolic murmur is heard at the aortic area and may be transmitted as far as the apex. There are also accessory signs of aortic insufficiency, such as Corrigan pulse, capillary pulse, large pulse pressure, etc. If the aortitis has involved the adventitia, where the aorta touches the pericardium, there will be a pericardial friction-rub.

Other signs are those of aneurysm, these depend on the location of the aneurysm. It is the most frequent mediastinal tumor, and may have no signs whatever. If it is in the ascending aorta, pulsation may be evident at the right first, second, or third spaces. You may feel a shock, diastolic in time, or a systolic thrill, or both. The circulation in the deep veins may be impeded, so that the superficial ones become conspicuous as they cross the chest. The aneurysm may press on the recurrent laryngeal nerve, in which case you will find the vocal cord paralyzed on that side, with resulting hoarseness or even aphonia. If the aneurysm irritates the sympathetic ganglia, you will find pupillary inequality and perhaps also anhidrosis. An aneurysm which presses sufficiently on the main branches of the respiratory tree will give you a tracheal tug, or the pulsations may cause a wavy respiration to be heard over the trachea. It is quite easy to picture this. Aneurysm on the right side may cause unequal radial pulses.

If the pulsating tumor occurs in the transverse part of the aorta, it may point either toward the right or the left. It is more apt to point toward the left, and produce signs like those found in aneurysm of the first part of the descending arch. Aneurysm in the left side of the mediastinum may cause signs similar to those of aneurysm on the opposite side. An expansile tumor will cause pulsation, diastolic shock, and systolic thrill, with dulness over this area. Aneurysm sometimes causes sys-

toxic and diastolic to-and-fro murmurs. On the left side aneurysm may press on the recurrent laryngeal nerve, perhaps causing hoarseness or aphonia in a similar way to mitral stenosis (when a large dilated auricular appendix with clot in it is sometimes found). The left pupil may be dilated, and there may be unequal pulses.

An aneurysm of the descending arch may point back in the left interscapular region, or near the second and third dorsal vertebræ, and gradually push through the tissues of the back. It may cause a pulsating tumor with signs like those of the other pulsating tumors. It also may erode the vertebræ, and cause transverse myelitis and paralysis. It has at times been the seat of wiring operations, the wires being run into the aneurysm through a hollow needle, and clotting promoted by an electric current. That method is not much used since the introduction of antisypilitic treatment for these patients.

Aneurysms occur occasionally in the abdominal aorta.

In addition to these physical signs, an x-ray taken with the tube about 6 feet away from the patient's chest is of value. I have some x-rays here (Figs 81-83). In this case the heart is slightly larger than normal, particularly on the left side. Also there is a wide area of shadow over the aorta. This measures about 7.2 cm. at the level of the second rib. The normal measurement is 5 to 7 cm. In case of a wide aortic shadow, one should recall that, anatomically, the aortic arch is on the bias, running from before backward, and from right to left.

Patients who have a purely syphilitic myocarditis have cardiac pain and sometimes precordial tenderness. Patients with palpitation, which at times is due to cardiac arrhythmia, not infrequently have an area of hyperesthesia in the cardiac zone, they usually develop cardiac insufficiency, with not very long duration of life.

The Wassermann reaction is the last sign, and may or may not be positive. It has the value of a single symptom in the presence of a positive history. Even with a negative Wassermann, we treat the patient, if other signs point to the existence of syphilis. Necropsy has justified this, as it has repeatedly

revealed syphilis of the vascular system, even though the Wassermann reaction was negative

An important consideration is that syphilis of the central vascular system is a tertiary manifestation of a constitutional disease. Therefore, as soon as you find it, look for other signs of tertiary syphilis. They occur from the scalp to the soles of the feet, from alopecia to perforating ulcers at the other extreme. Particularly look for syphilis of the central nervous system. Forty per cent of patients with syphilis of the central nervous system have vascular syphilis, and about 20 per cent of those with vascular syphilis have syphilis of the central nervous system. By this we mean tabes dorsalis, paresis, cerebrospinal syphilis, etc.

Treatment—The active treatment falls into two subdivisions that for the heart, and that for syphilis.

Treatment of the Heart—This is primarily the prevention and active treatment of cardiac insufficiency. We provide patients regularly with the folders issued by the New York Heart Association. These stress avoidance of overexertion—gastro-intestinal, mental, and physical. We caution the patient not to overeat, not to get constipated, and not to become fatigued from straining at stool. Not only must overexertion be avoided but also prolonged exertion, mental or physical, or sudden exertion, such as running for a street car, or trying to pick up a trunk. Certain patients need digitalis, in such cases we give it to them in the ward, and to take home. However, until their optimal dose has been ascertained, they are observed at frequent intervals.

When cardiac insufficiency arises, it also must be treated. Some patients receive quinidin. Sometimes large doses of digitalis are necessary for the heart. Sometimes we place patients on a Karrell diet. Occasionally theobromin or caffeine derivatives, or "novasurol" are administered to reduce anasarca.

Treatment for Syphilis—The treatment for active syphilis is with mercury, arsphenamin, potassium iodid, and bismuth. Mercury is given intramuscularly, usually in the form of mercury salicylate. The first dose is 0.03 gm, the second is the same. Then, if no symptoms of mercurialism develop, the dose is doubled.

and 0.06 gm is given in oil. That is the equivalent of 1 gram a week. At the end of the sixth or seventh injection the patient is usually given an interval of six weeks' freedom from treatment. During the period of mercurial treatment he usually takes iodid of potassium. Start with a saturated aqueous solution of KI, 5 drops three times a day for the first week, taken in half a tumbler of water. At the end of the first week increase the dose 1 drop each day until the patient is taking 50 drops three times a day after food. After the dose reaches 10 drops three times a day the amount of water is increased to a whole tumblerful. Many patients are not able to tolerate 150 drops of the iodid a day. Some cannot take more than 30.

After an interval of six weeks without mercury patients receive neo-arsphenamin intravenously. They are instructed to take a saline cathartic the morning they are to receive treatment, or to take castor oil the night before, to eat their breakfasts at the regular hour, and to report at the hospital between 1 and 2 P. M. without any lunch, and having eaten nothing since breakfast.

For the instructions as to the administration of the neo-arsphenamin I would refer you to Dr. Fordyce's lectures in the Department of Dermatology and Syphilology. Briefly, the patients receive very small doses the first two times—no more than 2 decigrams the first two injections. If this is tolerated well, with no reactions, increase to 3 or 4 and to 6 or 8 decigrams, given intravenously. During the first course of arsphenamin the dose is gradually increased as just described, but we never give more than 8 decigrams at any one treatment. After an interval of six weeks the patient is again placed on a course of mercury, iodid being taken at the same time.

These patients should always see a dentist and have their teeth put in as perfect condition as possible. We supply them with a mouth-wash to use after meals and at bedtime during the administration of the mercury, it contains potassium chlorate. Any good mouth-wash, however, is satisfactory.

We have been using bismuth for some patients—for those who do not tolerate mercury or arsphenamin, or neither. We

find that the bismuth is a very satisfactory addition to the drugs available for antisyphilitic treatment. At first we used a suspension of metallic bismuth prepared here at the hospital. Later we substituted ampules of bismuth potassium tartrate with butin. It is administered intramuscularly in the buttocks once a week. The technic is the same as for mercury salicylate.

Prognosis—There is no definite statistical evidence regarding the prognosis in this disease. It is, however, a very unhappy one. The disease occurs in the later part of life, the largest incidence being between thirty-five and fifty years. Coincident atheroma and arteriosclerosis are not unusual. Hence it is evident that the prognosis is poor. Patients seldom live more than five years after the disease is discovered, although they may live eight or nine.

SUGGESTED READING

Excellent reading on this subject is to be found in Sir Clifford Allbutt's *Diseases of the Arteries, Including Angina Pectoris*, 2d volume, and in *Syphilis du coeur et de l'aorte*, by Brin and Giroux.

CLINIC OF DR ROBERT F LOEB

PRESBYTERIAN HOSPITAL

THE DIAGNOSIS OF ACUTE RHEUMATIC PERICARDITIS

INCORRECT medical diagnosis results usually from one of two outstanding causes: either the case does not conform with the classical disease picture associated with a given condition or error results from carelessness on the part of the physician. Errors resulting from the first of these causes are in a measure pardonable, yet the number of these mistakes may be greatly diminished the more we extend our knowledge beyond textbook descriptions. Medicine would, indeed, be a very simple art were it not for the fact that vast numbers of patients present atypical symptom complexes in their diseases. Not infrequently their conditions are recognized only by clinicians who in their practice and in their study have encountered many of these aberrant forms. Blunders resulting from an incomplete history or an incomplete physical examination, or from inadequate laboratory investigation are, of course, inexcusable.

The diagnosis of acute rheumatic pericarditis is almost always a relatively simple matter on the basis of the history of the symptoms and the physical signs. In the case presented in this clinic the course of the disease and the physical signs were so unusual that even though the true condition was suspected at the time of admission, the correct diagnosis was not made and an unnecessary laparotomy was performed.

CASE REPORT

J P, an American boy of sixteen, whose occupation was that of a clerk, was brought to the hospital complaining of abdominal pain of nine days' duration. His family history was

negative His general health had been quite good in the past He had had measles, mumps, and chickenpox in early childhood There was no history of tonsillitis, scarlet fever, chorea, or rheumatism He gave no history of symptoms suggestive of failure of the circulatory system His respiratory history was negative For the past year he had suffered at irregular intervals from "indigestion," consisting of epigastric distress and the belching of gas after meals There was no history of real abdominal pain His bowels had been regular and his stools were apparently normal He gave no history of vomiting The remainder of his past history was irrelevant

Present Illness—Two weeks before admission he developed a sore throat which, however, did not keep him from his work Following this he developed a dry cough, which was still present at the time of admission Nine days before admission, while walking to work, he developed, rather suddenly, a pain in his epigastrium and vomited on the street He returned home and remained quiet, but not in bed, without any particular change in symptoms except for some variation in intensity of the pain, which occasionally kept him awake at night He vomited occasionally without any relation to his eating About a week before admission he developed slight dyspnea on walking and was considerably bothered by his dry cough He was not sure whether or not he had had any fever On the day of admission and on day of onset of his illness he noticed a blotchy rash on his arms At 3 A M on the morning of admission he was suddenly awakened from his sleep by a marked exacerbation of the pain in his epigastrium, for the relief of which he walked about doubled up and pressing on his epigastrium Respiration aggravated his pain He was seen by a local physician, who sent him to the hospital with a diagnosis of perforated gastric ulcer The history was not entirely reliable because of certain contradictory statements made by the patient and his family

Physical Examination—The patient looked acutely ill, his face was pale and sweating, his respirations were shallow and associated with a grunt which he attributed to the aggravation of the pain in his epigastrium with inspiration His lips were

dry, his tongue heavily coated, his teeth and throat appeared normal. His chest expansion was equal on both sides, his diaphragms were both high and moved but little with inspiration. His breath sounds were normal and no râles were heard. His heart appeared moderately enlarged to the right and left. The apex impulse was not seen or felt. The sounds were regular, slow, and slightly distant in quality. There was a soft systolic murmur at the aortic area which was thought to be organic rather than functional in nature. No friction-rub was heard in the supine, erect, or left lateral position. His pulses were equal, regular, slow, and of good quality. His abdomen was slightly full and showed no respiratory movement. There was "board-like" spasm down to the umbilicus, but below this his abdomen was relatively soft. There was marked tenderness on pressure and on the lightest percussion. Rebound tenderness was striking. Liver dulness was normal, but percussion over the liver caused pain referred to the epigastrium. There was no evidence of free fluid in the abdomen and there was no costo-vertebral tenderness. The remainder of the physical examination was negative except for a fading, blotchy erythema on the right forearm. Temperature 102.4° F, pulse 78, and respiration 30.

Laboratory Findings—White blood-corpuscles, 13 000, polymorphonuclears, 81 per cent. Fluoroscopic examination of the chest revealed the diaphragm high and almost fixed. The heart was moderately enlarged, suggesting the presence of mitral and aortic disease.

Course—After examination of the patient by both medical and surgical members of the staff, it was thought that, in view of the history of indigestion for a year, the marked exacerbation of pain in the abdomen a few hours before admission, and the classical signs of peritoneal irritation, that the most probable diagnosis was that of perforated peptic ulcer.

It was believed that the patient had a chronic heart lesion of rheumatic origin, but in the absence of a pericardial friction-rub, joint pains, or evidence of pericardial effusion by x-ray, a diagnosis of pericarditis did not seem warranted. There was no

reason to suspect occlusion of a coronary artery, though the distribution of pain in this condition is not infrequently abdominal. A laparotomy was performed and the peritoneal cavity was found to be normal.

The morning following operation a soft diastolic murmur was heard at the apex and systolic adventitious sounds appeared just to the left of the sternum suggesting a beginning pericardial rub. A pleural rub developed on the right below the nipple. The second day after operation there was a loud "to-and-fro" pericardial rub heard over the greater part of the precordium. This rub and the pleural rub persisted for a week and a small amount of fluid developed at the right base. The patient's temperature varied irregularly between 98° and 103° F for three weeks and then returned to normal. *At no time did the patient have any precordial or chest pain.* There were no joint pains at any time during his illness. Two years after this attack the patient is perfectly well except for some cardiac enlargement and evidence of mitral insufficiency.

DISCUSSION

While this case seemed quite unusual, a search of the literature revealed the fact that Holden¹ has reported 3 cases of pericarditis in which the early symptoms were referred entirely to the abdominal cavity. One of these was a case of purulent pericarditis, which began with a sudden abdominal pain, and forty-eight hours after a laparotomy the pericardial rub developed. The other 2 cases reported by Holden occurred in children of four and ten years and were presumably rheumatic in origin. The pericardial rub appeared twenty-four and forty-eight hours after operation, respectively. There is no mention of precordial pain in any of these cases.

In order to ascertain the relative frequency of the various signs and symptoms of acute rheumatic pericarditis, a study was made of the records of 34 patients suffering from this condition in the wards of the Presbyterian Hospital during the past few years. While this series of cases is not very large, the constancy

¹ Holden, W. B., Northwest Medicine, 1920, vol. xiv, p. 230.

of certain symptoms and signs is rather significant. Analysis of these cases yields the following results:

1 *Age Incidence*—About 60 per cent of the cases occurred between the ages of ten and twenty years. Only 1 case occurred in a person over thirty years.

2 *Previous Cardiac or Rheumatic History*—This was present in 77 per cent of the cases studied.

3 *Mortality*—About 35 per cent of the cases died during the attack of pericarditis. Most of the deaths occurred in patients presenting an old history of cardiac disease.

4 *Fever*—All of the 34 cases of this series had fever higher than 100° F (by rectum) and more than 90 per cent had temperature over 101° F. The duration of the fever was extremely variable.

5 *Cough*—This symptom was present in 100 per cent of the cases and was usually of the unproductive irritative type.

6 *Pericardial Friction-rub*—This sign was naturally present in all of the cases because the diagnosis was ultimately made up on its appearance. The time of appearance of the friction-rub in the course of the rheumatic disease was found to vary considerably. Pericarditis was, however, the first manifestation of rheumatic disease in a few cases.

7 *Dyspnea*—This symptom was present to a variable degree in 86 per cent of the cases.

8 *Precordial pain* was present in about three-fourths of the cases and was present in *all* of the cases without previous history of pericarditis. Of the 8 cases having no precordial pain, 6 were in children under ten years of age, and pain localization was probably inaccurate. It is interesting to note that in the case presented in this clinic, as well as in Holden's patients, there was no precordial pain in spite of the fact that the patients were apparently suffering their first attack.

9 *Rheumatic pleurisy*, as diagnosed by the presence of a friction-rub or by the demonstration of an effusion by x-ray or by thoracentesis, was present in 20 or about 60 per cent of the cases. Seventy per cent had signs of compression, fluid, or consolidation at the angle of the left scapula.

10 *Joint pains* occurred in only 60 per cent of the cases and in less than half of these patients were they recorded as being severe

11 *Abdominal symptoms* were present in 5 cases, or 15 per cent, of the series. These symptoms were recorded either as epigastric pain, "distress," vomiting, or of a combination of all of them. The pericardial rub appeared fourteen, eleven, and four days after the onset of abdominal symptoms in 3 of the cases. In the other 2 cases the rub was present at examination seven and three days, respectively, after the onset of the abdominal symptoms. In no case was the abdomen described as being rigid.

12 *Leukocytosis* was present twenty-eight times. It is rather surprising that 6 of the 34 patients should have had no leukocytosis throughout the course of their disease. In 60 per cent of the cases the white count was between 10,000 and 20,000. A marked increase in the number of polymorphonuclear cells was unusual.

13 *Secondary anemia* as judged by the ordinary clinical methods of hemoglobin determination was present in 86 per cent of the cases. Only those patients with a hemoglobin below 80 per cent were considered as having an anemia.

14 *Pulse and respiration* rates were accelerated to a variable degree in most of the patients.

15 *Electrocardiographic examinations* were made in 17 of the cases studied. Of these cases all but one had some deviation from the normal. Fifty per cent of the changes were very marked.

From this case analysis it may be seen that the most characteristic signs and symptoms of acute rheumatic pericarditis are fever, cough, dyspnea, moderate leukocytosis, and precordial pain, and that these symptoms, as well as abdominal distress, may precede the appearance of the pericardial friction-rub. The history of a previously damaged heart and the presence of a group of these symptoms should lead to more than a suspicion of the diagnosis.

CLINIC OF DR ARTHUR E NEERGAARD

PRESBYTERIAN HOSPITAL

MENINGOCOCCUS BACTERIEMIA

WE wish to discuss with you this afternoon, gentlemen, 2 cases of meningococcus bacteriemia without demonstrable localizing lesions

In July, 1911 Cecil and Soper reported a case of meningococcus sepsis with meningococcus endocarditis proved by necropsy, and suggested that this might throw some light on the etiology of meningococcus meningitis as a blood-stream infection

Further developing this idea in April, 1919, as the result of his work on meningococcus infections in the U S Army, Herrick reported a series of cases whereby he showed that a blood-stream infection was present in many cases prior to the manifestations of meningeal involvement, and that in some cases this latter stage is never reached

He cites 6 cases 1 and 2 Fulminating sepsis with positive blood-culture and no meningeal or other focal symptoms on necropsy 3 A case similar to those we are about to present, onset with tonsillitis, followed by polyarthritis, recurring chills and irregular fever, rapid anemia, maculopapular eruption, not unlike that of typhoid fever, positive blood-culture, no relief from salicylates, but yielding rapidly to antimeningococcus serum 4 Prolonged phase of sepsis with irregular fever and positive blood-culture, eventually developing a meningitis 5 Meningococcus pleurisy 6 Rash suggesting measles, acute nasal sinusitis, and positive culture from sinus pus

The above cases were collected during an outbreak when any soldier with a fever was investigated from the standpoint of a possible meningococcus infection Our 2 cases took us quite unawares, and, as will be seen, the correct diagnosis was in no way suspected at the onset

Case I—E W, female, thirty-five, married American housewife Admitted September 9, 1918, complaining of chills and fever and pains in legs for nine weeks

Past History—Previous health and habits good "Diphtheritic sore throat" eight years ago Pneumonia six years ago Some ovarian trouble four years ago

Present Illness—Nine weeks ago, sudden onset of chills and fever, coming on in the early evening Awoke next morning to find that ankles and knees were stiff and painful on motion This disappeared during the day Took quinin for a few days, without relief Since this onset has had chills and fever every day, except one, in the late afternoon or early evening No sweats Has continued to have stiffness of legs, arms, shoulders, and hands, and sometimes shooting pains in the legs lasting for part of a day These are followed at times by the appearance on legs or arms of small red spots definitely tender Patient has been in bed for the most part since onset and has lost 15 pounds Appetite remains good and bowels are regular

Physical examination showed a poorly nourished and nervous woman Scattered over her extremities were numerous slightly elevated erythematous areas about $\frac{1}{4}$ inch in diameter Pupils normal Fundi normal Pharynx slightly congested No cervical rigidity. Lymph-nodes not enlarged Heart normal Blood-pressure 110/75 Lungs clear Abdomen Right kidney palpable Spleen not felt Knee-jerks hyperactive Pelvic negative Wassermann negative

September 9th—Blood-culture negative

September 10th—White blood-corpuscles 14,000, polymorphonuclears 70 Urine negative for pus Blood smear negative for malarial parasites

September 11th—Blood negative for gonococcus and tubercle fixation Widal negative 1 10 to 1 320 Chill in afternoon with rise of temperature to 101.6° F

September 13th—Throat culture No K L Streptococcus present

September 16th—Repeated searches for malaria negative

September 26th—White blood-corpuscles 19,000, polymorphonuclears 78, hemoglobin 70, red blood-corpuscles 4,128,000

September 28th—Painful joints and painful nodules on legs persist

October 10th—White blood-corpuscles 12,400, polymorphonuclears 71 Blood-culture showed Gram-negative diplococci in both flasks

October 15th—Blood-culture Gram-negative diplococci both flasks, morphologically and culturally like meningococci, but not agglutinated by meningococcus polyvalent serum

October 18th—Lumbar puncture clear fluid, normal pressure, 70 cells, mostly lymphocytes Globulin negative Culture contaminated

October 19th—Following lumbar puncture had headache and stiff neck for several days

October 22d—Culture of nasopharynx—no meningococci *Bacillus influenzae* present Blood-culture still positive

October 27th—No agglutination of organism by either Board of Health or Rockefeller Institute antimeningococcus serum nor by patient's own serum in dilution 1 to 5 The organism grows best in blood-broth and on blood-agar, poorly on ascitic agar, not at all on plainer media In spite of failure to agglutinate, the bacteriologists were convinced that the organism was a meningococcus

October 28th—Normal horse-serum (amount not stated) injected intravenously

October 31st—Blood-culture—no growth White blood-corpuscles 7800, polymorphonuclears 62

November 1st and 9th—Agglutination against patient's own organism—negative

November 5th—Blood-culture—no growth

November 11th and 12th—Throat culture negative for meningococci

Temperature chart

September 9th—Spike to 102 8° F

September 10th—Normal

September 11th—Spike to 101 6° F

September 12th —Normal

September 13th —Spike to 101° F

September 14th — 100° F

September 15th — 101.6° F

September 16th —Normal

September 17th — 102° F

September 18th —Normal

September 19th — 102° F

September 20th — 102.2° F

September 21st — 103.4° F

September 22d to 29th —Not above 100.6° F

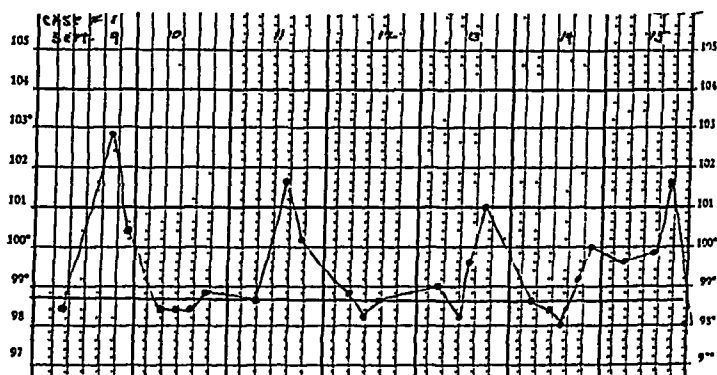


Fig 84

September 30th — 102° F

October 1st —Normal

October 2d — 102.4° F

October 3d — 102.4° F

October 4th — 104.2° F

October 5th —Normal

October 6th — 103.6° F

October 7th — 103.6° F

Last spike *October 26th*

From then normal till discharge

A graphic record of the temperatures from September 9th to 15th is shown to illustrate the irregularity of the fever

Gradual disappearance of the stiff joints and eruption

Discharged November 13, 1918, one hundred twenty-nine days after onset

Seen January 22, 1919 Perfectly well

Case II—J H, fifteen, male, single Sent to the hospital December 18, 1924 by his doctor, with a diagnosis of typhoid fever No history of any symptoms except that seventeen days previously he had vomited once, and had had some fever off and on since, but had been up and about The only abnormal findings on physical examination at that time were Temperature 99.8° F, huge red tonsils, slight general glandular enlargement, and white blood-corpuscles 13,400, polymorphonuclears 77 He was told to take a cathartic and some prescribed grippe capsules, to remain in bed for two days, and to return for observation in four days to the Out-patient Department

Returned as instructed on December 22, 1924

Chief Complaint—Pain in legs and back of neck

Family History—Negative

Past History—Habits good General health good Small-pox as child No other acute illness

Present history dated back two or three weeks, when he began to have headaches, which have disappeared Then he developed a rash which has been intermittent since Then he developed some pain and swelling of one ankle His doctor at one time found his temperature 103° F Patient would stay in bed in the morning, but insisted on going out in the afternoon Finally, as above stated, his doctor sent him to the hospital

Physical Examination—Temperature, 100.6° F; pulse, 104. Looks sick Eyes somewhat puffy No conjunctival hemorrhages Tonsils very large and red, no exudate Rather marked enlargement of superficial lymph-nodes, including the right epitrochlear Skin showed a fading, pale red, maculopapular eruption over trunk, especially the back, and to less extent on face and extremities Heart and lungs negative Spleen just palpable Reflexes normal No cervical rigidity, Kernig, or Babinski White blood-corpuscles 20,300, polymorphonuclears 79, lymphocytes 19, eosinophils 2

He was seen at this time by several of us and the diagnosis was not clear. The eruption suggested to some a fading German measles, to others secondary lues, but with his red throat, story of fever and joint involvement, he was admitted to the hospital as a probable rheumatic fever with erythema multiforme. The patient was of a low-grade mentality and at all times unco-operative and never gave exactly the same history on any two occasions.

December 23, 1924—Hemoglobin 90 per cent, red blood-corpuscles 4,540,000, white blood-corpuscles 19,000, polymorphonuclears 79, lymphocytes 12, large mononuclears 7, transitionals 2. Wassermann negative. Widal and Parawidal negative. α -Ray of sinuses negative.

December 27th—Blood-culture, no growth.

December 24th—Electrocardiogram. P notched in all leads. T upright throughout. Definite notching of P in all leads suggests mitral stenosis.

Electrocardiograms repeated January 9, 1925, January 30, 1925, and February 6, 1925 showed this notching much less marked with no evidence of mitral stenosis.

January 2, 1925—White blood-corpuscles 22,400, polymorphonuclears 87.

January 7, 1925—Blood-culture—no growth.

January 11th—Blood-culture. Gram-negative diplococcus in both flasks. This organism was agglutinated by New York State antimeningococcus serum.

January 13th—Throat culture. Numerous Gram-negative diplococci found.

January 15th—Patient's organism was tested by Shibley's method to determine to what extent its electrical charge and agglutination were affected by antimeningococcus serum, with results as follows:

N Y State antimenin-				
gococcus serum	1-40 P D =	- 6 2 MV,	agglutination complete	
B of H antimeningo-				
coccus serum	1-40 P D =	- 14 6 MV,	"	+
Normal horse serum	1-40 P D =	17 1 MV,	"	none
No serum	1-40 P D =	38 8 MV,	"	"

January 16th—Was found slightly sensitive to horse-serum. Was desensitized and received 177 c.c. antimeningococcus serum intravenously. No reaction except nausea and some urticaria.

January 18th—Temperature spike to 104° F.

January 19th—Blood-culture—no growth. Received 100 c.c. serum.

January 20th—Blood-culture—no growth. Received 100 c.c. serum.

January 21st—Blood-culture showed meningococcus which agglutinated 1:80, 100 c.c. serum given.

Patient had now had 477 c.c. serum without, so far as we could see, any definite effect on his infection. So no more was given.

Blood-culture on January 23d still positive. This was the last positive culture.

January 29th—Hemoglobin 60, red blood-corpuscles 3,060,000.

January 31st, February 13th, February 21st—Blood-culture, no growth.

February 7th—Direct transfusion of 800 c.c. blood without reaction.

February 10th—White blood-corpuscles 7000, polymorpho-nuclears 62.

February 18th—Tonsillectomy.

His temperature as shown by Fig. 85 showed irregular spikes with afebrile periods on one occasion as long as four days. The last spike was on February 5th, after which his temperature was never above 100° F., and gradually became normal.

At no time did he manifest signs of meningeal involvement. For this reason lumbar puncture was purposely omitted to avoid the possibility of infecting his supposedly sterile meninges with blood known to contain meningococci. The patient was discharged cured on February 26, 1925, and has remained perfectly well ever since. He was last seen on April 7, 1925.

These 2 cases have been presented in perhaps undue detail in order that this clinical picture may stamp itself upon our minds. If we study them carefully, we find that both present the following striking features:

1 The correct diagnosis was neither established nor sus-

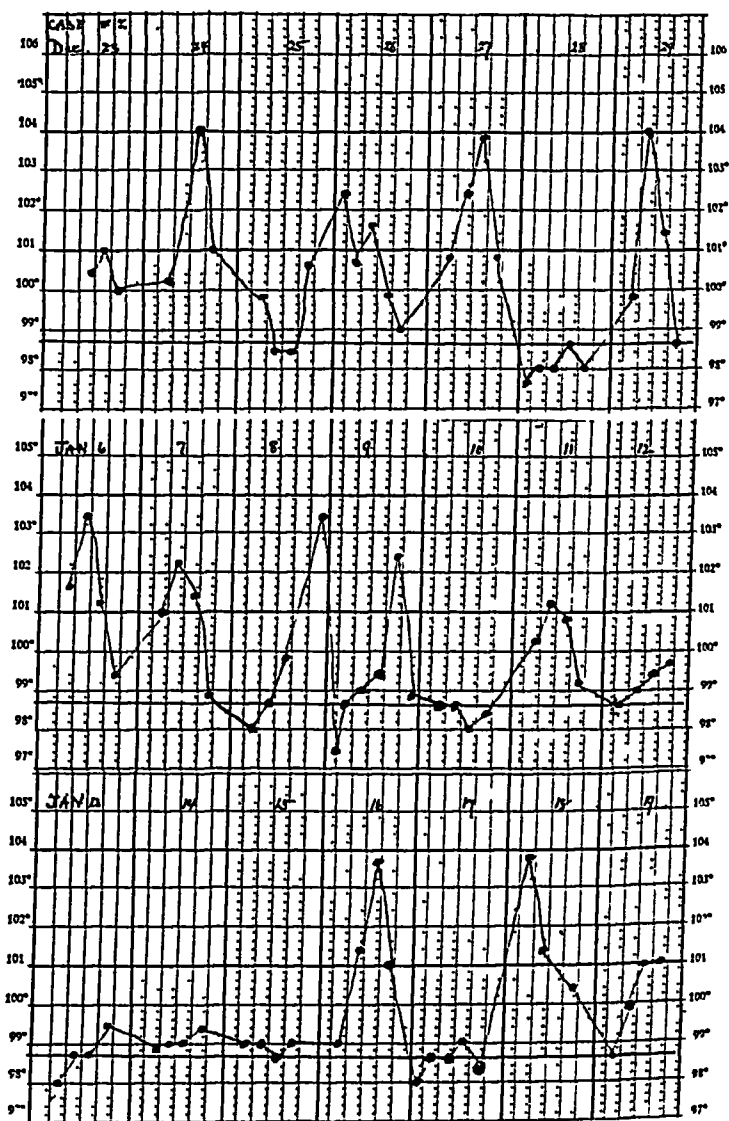


Fig 85

pected until the cases had undergone careful study for a week or

more At the onset the diagnoses of typhoid, influenza, malaria, septicemia, and acute rheumatic fever have been suggested—only in turn to be rejected

2 The recovery of Gram-negative diplococci from the blood-stream by the use of special media, such as blood-broth or blood-agar The failure of this organism to grow in the glucose broth ordinarily used for routine cultures clouds the diagnosis Where this condition is suspected, cultures should be taken on the special media mentioned

3 A temperature curve which shows spikes occurring with no regular sequence, coming sometimes every day for several days, at others with an intervening afebrile period of from one to four days If the patient first consults the doctor on an afebrile day and no malarial parasites are found in the blood, the natural tendency of the doctor would be to discount the patient's story, especially as these patients, except when the fever was at its fastigium, did not appear or feel particularly sick.

4 An eruption usually maculopapular, sometimes hemorrhagic, simulating the rash of typhoid, measles, or the toxic erythemas It has been diagnosed as a drug rash and as erythema multiforme

5 Fleeting joint pains

It seems probable that these cases are more common than we suspect, and that if we bear this clinical entity in mind a little watchful waiting may divert more cases from the discard of fever of unknown origin to be classified in this category. It is with this hope in view that these cases are presented to you

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CLINIC OF DR ALVAN L BARACH

PRESBYTERIAN HOSPITAL

THE THERAPEUTIC USE OF OXYGEN IN ACUTE RESPIRATORY DISTURBANCES

OUR object in this clinic is to present the subject of oxygen therapy from several points of view

- 1 Physiologic basis of the therapeutic use of oxygen
- 2 Efficacy of the methods employed
- 3 Illustrative cases
- 4 General discussion of the results of oxygen treatment.

Although the use of oxygen is an old procedure in clinical medicine, knowledge of the physiology of oxygen want as well as of adequate methods of combating it has been developed largely as a result of modern investigation. As a consequence we are at the present time in the process of constructing a new conception of a remedy that in the past was both poorly understood and poorly applied.

We shall first consider the effects of a diminished oxygen supply in a normal individual. Evidence bearing on this question has been derived from ascent to high altitudes, in which there is a progressive lowering of the partial pressure of oxygen in the air breathed, from experiments in chambers in which the concentration of oxygen is artificially lowered, and from carbon monoxid poisoning. In all those instances symptoms of oxygen want appear, varying with the suddenness with which oxygen is decreased, the extent to which it is decreased, and the length of time oxygen want is endured. In abrupt severe exposure to a low oxygen environment unconsciousness may appear without premonitory symptoms.

In gradual exposure to low oxygen, as in mountain sickness,

a feeling of exhalation is apt to be replaced in six or eight hours by headache, lassitude, loss of appetite, nausea, and often vomiting. The lips are blue, the pulse is rapid, there is a sense of oppression in the chest, dyspnea on slight effort, muscular weakness, and sometimes complete prostration. On the second day the eyes are heavy and dull, the tongue furred, and the bowels disturbed. Powers of memory and sane judgment are impaired, and anoxic persons become subject more or less to irrational fixed ideas and to uncontrolled emotional outbursts. This malady befalls some individuals at a lower, others at a higher, altitude, but for all there is a critical elevation beyond which escape is impossible. In chamber experiments by Haldane, Barcroft, Schneider, and others these effects have been duplicated and shown to be due purely to oxygen want. Haldane became irrational in a chamber experiment in which the atmospheric pressure was lowered to 350 mm. Barcroft lived for six days in a chamber in which the partial pressure of oxygen fell to 84 mm. The oxygen saturation of his arterial blood was 88 per cent and after exertion 83.8 per cent. He lay in the chamber, racked with headache, with occasional vomiting, his pulse approximately 30 beats above normal, and at times able to see clearly only as an effort of concentration. He became faint on exertion.

These unpleasant and harmful effects are produced in a normal individual by a degree of oxygen want that is of not infrequent occurrence in pneumonia. The measurement of the arterial oxygen saturation introduced by Hurter and Stadie was followed by the demonstration that severe oxygen deprivation may occur in lobar pneumonia. In other words, mountain sickness may be an additional burden to the toxemia of pneumonia. Furthermore, more severe harm may result in the way of delirium, unconsciousness, cardiac and respiratory embarrassment, if we are able to apply the effects on normal individuals to patients with pneumonia who suffer from a similar degree of anoxemia.

With severe and prolonged exposure to want of oxygen the after-symptoms are of an extremely formidable nature, involving the nervous system, the circulatory and respiratory systems.

In cases of carbon monoxid poisoning consciousness may not be recovered, although within an hour or two after exposure to fresh air the blood may be almost normally saturated with oxygen. The patient may linger on for weeks in a comatose condition, with spastic muscles and occasional epileptiform seizures. In those cases in which the prolonged exposure to want of oxygen has been accompanied by muscular exertion the after-symptoms may be mainly cardiac. The pulse is feeble and irregular, the heart dilated, with a blowing systolic murmur; muscular exertion produces collapse.

The immediate cause of death is frequently failure of the respiratory center. With anoxemia of moderate severity the symptoms known as fatigue of the respiratory center make their appearance, namely, diminishing depth with increasing rate of respiration. In the severe cases the depth continues to diminish without compensation from the rate and the condition rapidly becomes dangerous.

The early recognition and treatment of anoxemia in carbon monoxid poisoning is thus a matter of great importance. Henderson has devised an apparatus in which carbon dioxid and oxygen are breathed in order to remove as quickly as possible the oxygen want of which the patient suffers. All these considerations apply to a varying degree to clinical cases of anoxemia; oxygen treatment in severe cases will not immediately dispel the symptoms which have been produced by oxygen want, although the anoxemia itself is relieved. The prevention of severe anoxemia can be accomplished only by early and adequate treatment.

In acute conditions cyanosis is an approximate measure of the degree of anoxemia, in cardiac disease more marked in the lips, and in respiratory disturbances in the nail beds. A leaden gray color in pneumonia frequently indicates severe oxygen want. Anemic individuals may fail to show cyanosis in the presence of severe anoxemia, due to the small amount of available hemoglobin. In chronic cases of disease cyanosis may exist without harm because of adaptive changes within the organism. Thus, cyanosis may have no relation to physiologic anoxemia, which

should be understood as a diminution in the amount of *free* oxygen in the circulating capillary blood. A further example may be found in the phenomena which attend increased breathing. Carbon dioxide is washed out of the blood and the oxygen saturation of the arterial system is slightly increased. Nevertheless, symptoms of anoxemia result, which are removed when pure oxygen is breathed. In other words, as discovered by Bohr, deficiency of carbon dioxide causes the hemoglobin to hold on more tightly to oxygen. This effect has subsequently been shown to depend on increased alkalinity. The importance of the Bohr phenomenon clinically is probably not great, although it illustrates the fact that an increased oxygen content of the blood may occur in the presence of physiologic anoxemia. In general, however, oxygen want is accurately determined by the oxygen saturation of arterial blood since this indicates the pressure at which oxygen is available to the tissues.

The hemoglobin of the arterial blood is normally 95 per cent saturated with oxygen. In pneumonia the arterial blood may show normal oxygen saturation, *i. e.*, 95 to 100 per cent, mild anoxemia, *i. e.*, 85 to 95 per cent, moderately severe anoxemia, 80 to 85 per cent, severe anoxemia, 70 to 80 per cent, and very severe anoxemia, *i. e.*, 60 to 70 per cent saturation. Through the employment of effective methods of administering oxygen Meakins, Barach and Woodwell, and Stadie have shown that arterial anoxemia in pneumonia can in the majority of instances be restored to, or near, the normal value and that, concomitant with the elevation in arterial oxygen, clinical betterment usually occurs.

If our train of reasoning is correct, namely, that harmful effects result from a diminution of the oxygen of the arterial blood, that such a diminution occurs in clinical disease, and that it is capable of relief by adequate methods of administering oxygen, we may now consider the most effective means of combating anoxemia in acute respiratory disturbances.

In a recent study we considered in some detail the effectiveness of various methods of giving oxygen, based largely on the concentration of oxygen in the inspired air. Under ordinary

conditions the oxygen concentration of the inspired air is 21 per cent. When oxygen is administered at a concentration of less than 30 per cent. little benefit is generally observed. Animal experiments indicate that air mixtures containing more than 70 per cent oxygen produce a serous pneumonia when respired continuously over periods of one to five days. The optimal therapeutic concentration of oxygen in the air breathed appears to be 40 per cent with a minimal and maximal range of effectiveness between 30 and 60 per cent.

The tube and funnel method, contributing 21 to 24 per cent oxygen in the inspired air, is practically useless.

The nasal catheter varies in effectiveness, depending largely on the flow of oxygen from the tank, but may be made a method of distinct value provided certain precautions are carried out. If 2 liters of oxygen per minute are discharged through the catheter, the oxygen of the inspired air may be between 30 and 35 per cent, depending on the tidal volume and the respiratory rate, factors which obviously vary the dilution of the oxygen admitted during the inspiratory phase. If 1 liter per minute is administered, 25 to 27 per cent oxygen is present in the inspired air.

The nasal catheter should be passed into the nasopharynx until it touches the posterior wall, then withdrawn $\frac{1}{2}$ inch and fixed to the forehead with adhesive. The terminal 1 inch of the catheter has four small holes instead of one in order to permit the relatively high flow of 2 liters per minute without discomfort. It must be recognized that catheter oxygen treatment causes enrichment of the inspired air only when nose breathing is present. For that reason small No. 10 French catheters are to be preferred, as they are less apt to cause mouth-breathing than large tubes which block the anterior nares.

We have found it advisable to use high pressure oxygen tanks instead of the low pressure variety in common use. Equipped with a reducing valve which is calibrated to deliver oxygen in liters per minute, the large high pressure oxygen tank maintains a constant rate of flow of 2 liters per minute for more than two days, whereas low pressure tanks have to be changed ten to

twenty times during this period. Furthermore, the cost of low pressure oxygen for hospital use is eight to ten times as expensive as high pressure oxygen, and even more costly in private practice.

If high pressure oxygen tanks are equipped with a rubber-wheeled truck they can easily be moved into the ward by nurse or orderly. The care of the reducing valve involves two simple but important precautions: (1) To insert no oil in the valve under any circumstances. (2) To turn on the oxygen to blow out the dust before attaching the valve. We have constructed

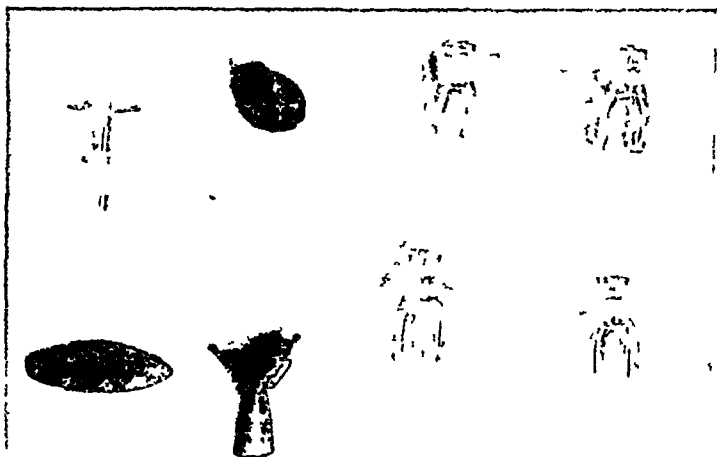


Fig. 86—Types of nosepiece and mouthpiece used in the rebreathing apparatus

an aluminum jacket with a front window of wired glass to fit over the valve. After attachment of the valve the jacket is slipped on and locked. The oxygen tank is placed in a special stand to prevent it from falling. In this way precautions may be taken against conceivable accidents involved in the use of high pressure oxygen. High pressure oxygen is available in 110 and 220 cubic feet sizes. The latter is well adapted to hospital use, the former to private practice.

For the mild or moderate cases of anoxemia in pneumonia the nasal catheter with an inlet of 2 liters oxygen a minute

is of distinct benefit, providing as it does approximately a 30 per cent concentration of oxygen in the inspired air. In severe anoxemia the catheter may be unable to produce the desired relief, and methods which yield higher concentrations of oxygen should be used if possible

More effective apparatus are those of Haldane and Henderson, but the employment of a mask in both render them rarely applicable because of the discomfort they produce

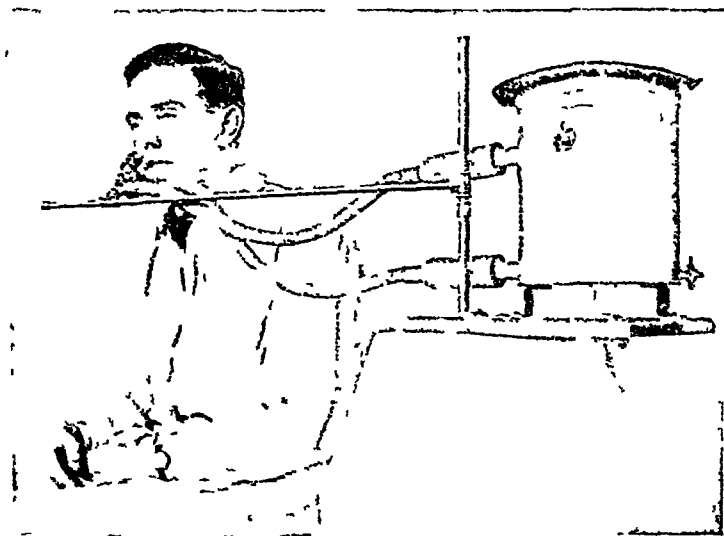


Fig 87 —Rebreathing apparatus with nosepiece in nostrils

The rebreathing apparatus which we first used five years ago relied on a mouthpiece of rubber or glass which was inserted between the teeth and lips of the patient, providing a rich oxygen mixture through the mouth and air through the nose. Although more comfortable than a mask it was frequently objected to when given over long periods. Recently we have substituted a glass nosepiece which fits in the nostrils. If one side of the nosepiece is closed by a small cork and the other fitted directly into the nostril or by the interposition of a 4-inch piece of rubber tubing of the same diameter, the patient breathes a rich oxygen

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mixture through one nostril and air through the other This provides approximately a 40 per cent oxygen mixture The CO_2 is absorbed by soda lime One liter a minute usually suffices to keep the rebreathing bag from collapsing (See Figs 86, 87 for recent nosepiece rebreathing apparatus and types of nosepieces and mouthpieces used)

For individuals who strenuously object to all forms of appliances to the face an oxygen rich atmosphere is desirable For this purpose oxygen chambers have been used by Stadie, Bar-



Fig 88 —Portable oxygen tent

croft, Binger, and Poulton Their rarity and expense make this form of oxygen treatment up to the present time limited

Barach and Binger have constructed a portable oxygen tent which possesses some of the advantages of a chamber inasmuch as the patient is enclosed in an oxygen rich atmosphere without any appliance to the face It has worked satisfactorily in the winter, but under warm conditions of the external atmosphere cooling is difficult (Experiments are being made to perfect this type of apparatus)

The head tent devised by Roth seems excellently adapted for the provision of an oxygen rich atmosphere without a direct appliance to the face

Davies has recently proposed a modification of the Haldane apparatus, employing a nosepiece which appears very effective

In administering oxygen we must bear in mind that one apparatus will not be suited to every patient. Frequently, the appliance to the face has to be varied to prevent discomfort. If mouth-breathing ensues, methods which depend on nasal respiration must be discontinued. At times we have used the nasal catheter, the rebreathing apparatus, and the portable oxygen tent in one patient. We are still engaged in the effort to find a simple portable apparatus which will be entirely comfortable and effective under all circumstances, although it seems likely that various types of apparatus will still be needed. The essential point is that effective methods be used. The patient is not the judge of his treatment in this instance any more than he is in other forms of therapy. In the relatively few cases in which subjective improvement is striking and immediate, the patient will demand the oxygen apparatus, but in the majority of individuals oxygen inhalation must be persisted in as long as cyanosis is present, notwithstanding the fact that the patient may notice no relief. As far as possible a method should be used which is effective and at the same time comfortable to the patient.

Case I—History—N. J., male, age sixty. Following a cold of two weeks' duration patient became acutely ill with cough, fever, and difficulty in breathing. Symptoms became progressively worse, and on the fifth day patient entered the hospital. Examination showed an old thin negro sitting propped up in bed, appearing very feeble, breathing rapidly, and with obvious distress. Lungs were dull over both lower lobes posteriorly with bronchial breathing and crepitant râles, more marked on the left side. Heart was not enlarged. No murmurs heard. Action was rapid and regular. Vessels palpably thickened. Pulse full and bounding. Abdomen distended, tense, and tympanitic. Liver palpable, white blood-corpuscles 16,500, polynuclears

92 per cent, red blood-corpuscles 3,690,000, hemoglobin 70 per cent. Urine had very faint trace of albumin and occasional granular cast. Sputum contained pneumococcus Type IV. Blood-culture sterile.

Course—During the next five days patient was given general medical treatment, including digitalis and oxygen through nasal catheter. At the end of this period he appeared very toxic, dyspnea and weakness being more pronounced than on admission. *x*-Ray of chest revealed mottled shadows in right lower lobe and a dense more diffuse shadow in left lower lobe. White blood-corpuscles were 51,000, polynuclears 95 per cent. Repeated sputum culture gave pneumonococcus IV, again with sterile blood-culture. Arterial oxygen saturation was 82 per cent, arterial CO₂ content 29.0 vol per cent (blood drawn with nasal catheter *in situ*). Temperature 102° F, pulse 100, respiration 40.

The patient was then put in the oxygen tent, the oxygen concentration being kept at 40 per cent, and remained there for seven days, for the most part twenty to twenty-four hours of the day. Oxygen administration on the first day in the tent, the tenth day of disease, was followed in three hours by a drop in pulse-rate of 12 beats per minute without change in temperature or respiratory rate. On the following date temperature fell, and patient seemed better. This lasted but one day, when temperature again mounted to 103° F, with increase of dyspnea and weakness. On the fifteenth day of disease his pulse, previously good, became thready, rate 120, and occasionally intermittent. Three unsuccessful exploratory chest punctures were made to discover a possible empyema which was suggested by the clinical course and by the *x*-ray films. Patient seemed hopelessly ill, respirations having mounted to 58. The arterial oxygen saturation out of the chamber was 78 per cent. The oxygen concentration was kept at 45 per cent overnight. On the following day temperature remained up, with, however, considerable improvement in his general appearance, a fall in respiratory rate to 48 and a better quality to his pulse, rate 110. Arterial oxygen saturation in chamber was 90 per cent. On the

next day, the seventeenth day of illness, the temperature dropped to 98.6° F, pulse to 90, and respiratory rate to 40. On the sixteenth day oxygen was stopped, temperature 98° F, pulse 86, respiration 28.

Two weeks after temperature was normal, the arterial oxygen saturation was 98 per cent, the arterial CO₂ content 43.3 vol per cent. It is also of interest that the disappearance of anoxemia under oxygen treatment was followed by a fall in circulating hemoglobin as measured by the oxygen capacity. The patient had a secondary anemia that probably contributed to the anoxemia of which he suffered.

Comment—This patient seemed desperately ill when oxygen treatment was begun. His prostration and weakness throughout the entire course of illness were marked. It appeared that the seven days which he spent in the chamber tided him over until he developed sufficient antibody. When out of the chamber on the fifteenth day for exploratory chest punctures and for x-ray of his chest his dyspnea was at its worst. Several hours later, breathing 45 per cent oxygen, there was noticeable improvement in his breathing and later in the night in his pulse. Oxygen by nasal catheter did not seem to be of much help, the arterial oxygen saturation being 82 per cent with catheter *in situ*, and on two occasions being 90 per cent in the chamber. It is of considerable interest that the arterial CO₂ content rose from 29 to 42.3 vol per cent. during convalescence, indicating a lowered bicarbonate content of the blood during the acute stage of the illness.

Case II—History—Man, age twenty years, suffered with severe abdominal pains for two days prior to admission to hospital. An acutely inflamed appendix was removed after dense adhesions were divided. Three days after operation he suddenly became intensely cyanotic, fighting and gasping for breath. His respirations rose to 38 and four hours later to 50 per minute. His pulse mounted immediately to 160. Temperature was 100.5° F. Oxygen administered through nasal catheter brought slight relief to his cyanosis. Pleurotomy of 500 cc was done.

without altering his condition. Four hours after onset of attack oxygen was administered by the mouthpiece rebreathing apparatus, patient inhaling between 50 and 70 per cent. oxygen. At the end of one hour pulse dropped from 160 to 140, respiratory rate from 50 to 40, and cyanosis largely although not entirely disappeared. The lips, face, and fingers developed a pink color, but of a darker hue than normal. The patient experienced immediate subjective relief, and subsequently demanded oxygen most of the time when he was not receiving it. He was given oxygen through the night, and on the following two days, one-half hour on, and one-half hour off. Twenty-four hours after onset of dyspnea patient developed a friction-rub in left axilla and coarse râles in right axilla. Two days later the picture was that of bronchopneumonia. Temperature was 103.8° F., pulse 126, respiration 32. Cyanosis and dyspnea continued, although much diminished. Oxygen was administered fifteen minutes every hour for the next two days, when temperature and pulse approached normal and oxygen was discontinued.

x-Ray examination showed a large shadow at base of left lung three days after pulmonary infarction took place. During the course of bronchopneumonia x-ray showed additional shadows at right base. Mottled shadows persisted in left base two months later, although smaller in extent than on first examination.

Comment—The case seems an evidence of an unusually large infarct of the left pulmonary artery and possibly, to a slighter extent, of the right pulmonary artery. Respiratory embarrassment was extreme. Adequate oxygen therapy produced striking improvement in circulation, respiration, general appearance, and patient's comfort, although symptoms and signs of respiratory embarrassment persisted to a considerable degree even during the plentiful inhalation of oxygen.

Case III—Male, age thirty-eight. Two and a half weeks after an operation for removal of appendix patient developed a cough, rusty sputum and a temperature of 104° F. Physical examination showed a thin adult man, in moderate dyspnea and slightly cyanotic. Lung signs indicated consolidation of right

lower lobe, white blood-corpuscles 15,800 Polynuclears 90 per cent. Sputum contained no pneumococcus Blood-culture sterile. On the fifth day of disease dyspnea, cyanosis, and prostration had increased and oxygen was given with the rebreathing apparatus, using the nosepiece Second sputum examination revealed pneumococcus Type I Blood-culture positive for pneumococcus Type I Serum treatment was begun On the sixth day consolidation spread to left lower lobe, right middle lobe, and right upper lobe, with no signs of resolution in right lower lobe. Patient grew rapidly worse He objected to the nosepiece, and oxygen by this method was discontinued One hour later arterial oxygen saturation was 73.8 per cent. Gurgling respiration had developed Nasal catheter for one hour at 2 liters per minute resulted in no perceptible change, arterial oxygen saturation with catheter *in situ* 68.5 per cent Patient was then put in oxygen tent, the concentration of oxygen being kept at 50 per cent. Four hours later decided improvement was manifest. His breathing was easier, nails were somewhat pink, his pulse had slowed and there was no gurgling in his throat. Serum treatment was resumed and on the following day temperature began to fall Arterial oxygen saturation in the chamber was 90.2 per cent Patient was kept in the tent in all three days, when he was removed convalescent. Subsequently, he was operated on for empyema

Comment—On the sixth day of disease this patient had a positive blood-culture, with the left upper lobe the only lobe uninvolved by consolidation His arterial oxygen saturation at its lowest was 68.5 per cent. He would tolerate no appliance of any portable oxygen apparatus The nasal catheter did not raise the oxygen content of the arterial blood The inhalation of 50 per cent. oxygen in the tent increased his arterial oxygen saturation to 90.2 per cent., and was followed by unmistakable clinical improvement During his residence in the oxygen tent massive serum treatment was followed by sterile blood-culture and crisis It seemed probable that oxygen treatment tided the patient over to a period when a specific immunity was established with the help of serum administration

Case IV—Female, age sixty-two Patient felt poorly for three days when she developed a frequent cough, with blood-streaked sputum and fever Entered the hospital on the following day Physical examination showed an old woman who appeared at least seventy years, in slight dyspnea, without cyanosis or prostration Lungs showed beginning consolidation of right lower lobe Heart was enlarged to the left, with numerous premature contractions Blood-pressure 185/80 General arteriosclerosis White blood-corpuscles 21,800 Polynuclears 84 per cent Sputum pneumococcus Type III During the next five days consolidation spread to right upper lobe, right lower lobe, and right median lobe, and edema of lungs began Blood-culture was positive for pneumococcus Type III

Arterial oxygen saturation was 72.6 per cent She was put in the oxygen tent, oxygen concentration being kept at 50 per cent On the following day arterial oxygen was 91.2 per cent Respiratory rate had decreased from 36 to 20, pulse from 110 to 100 Edema of lungs persisted, and patient seemed to grow steadily weaker On following morning she was taken out of the tent for a short time and deep cyanosis returned, with immediate acceleration of respiratory rate to 36 Replaced in 50 per cent oxygen, color returned and respiratory rate slowed The pulse-rate had gradually risen during the night to 160, at noon it became impalpable, and patient died

Comment—This patient was in the hospital from the first day of disease Although she gave her age as sixty-two, she seemed at least ten years older The invasive power of pneumococcus Type III in this instance could be watched from day to day, as consolidation involved progressively every lobe except one, with the organism present in the blood-stream The inhalation of 50 per cent oxygen slowed her respiratory rate to 20 and brought the arterial oxygen almost to the normal level Her pulse on the first day in the tent was 100 and of good quality Her toxic appearance, however, never left her, and her death occurred finally as a result of cardiac failure

DISCUSSION OF RESULTS

The 4 cases which have been presented illustrate some of the methods and results of the therapeutic use of oxygen¹ We shall briefly summarize the cardinal points which they bring out

The nasal catheter appears to be of distinct benefit in the treatment of mild and moderate cases of arterial anoxemia, provided 2 liters of oxygen per minute are discharged through the catheter For the relief of severe arterial anoxemia methods of greater effectiveness are commonly necessary The rebreathing apparatus offers a sufficiently high concentration for all degrees of oxygen want, and may be used with a mouthpiece or nosepiece Some patients object to any appliance to the face over long periods Under these circumstances intermittent oxygen inhalation may be resorted to If an oxygen chamber or tent is available the patient may breathe an oxygen-rich mixture without any restrictions incident to bedside methods

Of the clinical results, the clearing or diminution of cyanosis is the most constant Lessened delirium and increased rest are apt to follow In a certain number of cases there is partial relief of dyspnea Most patients appear more comfortable in an oxygen rich atmosphere, although the dyspnea itself may largely persist The pulse-rate is generally reduced The respiratory rate may or may not be lowered The arterial oxygen saturation is usually restored to or near the normal value by adequate oxygen treatment

Relief of dyspnea was most marked in the case of pulmonary embolus in which there was a sudden severe deprivation of oxygen Both pulse- and respiratory rate were substantially decreased Of the 3 patients ill with pneumonia, 2 showed striking clinical betterment following the inhalation of oxygen The fourth patient succumbed to a pneumococcus Type III lobar pneumonia with a bacteriemia of the same organism She was a woman approximately seventy years old who was observed from the first day of disease Her dyspnea was not marked The arterial anoxemia was relieved by oxygen treat-

¹ For more complete discussion of methods see *Methods and Results of Oxygen Treatment in Pneumonia*, by Alvan L. Barach, *Arch Int Med*, 1925

ment and the respiratory rate slowed from 34 to 20. Nevertheless, with advancing consolidation involving every lobe except one, she grew progressively weaker, became comatose, and died.

The evidence from the above cases and from others elsewhere reported indicates that oxygen therapy is of distinct value in acute respiratory disturbances characterized by severe dyspnea and cyanosis. The respiratory burden is relieved and the harmful effects of oxygen want prevented. In postoperative embolus resulting in marked cyanosis and dyspnea such aid may be crucial. In some instances in pneumonia the inhalation of oxygen appears to be responsible for tiding the patient over to a period of crisis. When, however, the clinical condition is largely the result of toxemia, especially when accompanied by a blood-stream infection, oxygen therapy is of little or no benefit.

The rapid respiration of pneumonia is generally not due to oxygen want, although it may contribute to it. In some cases the respiratory rate is distinctly slowed by the inhalation of oxygen, whereas in others it is unaffected. The factors which govern this response are undetermined. From an empirical point of view one might say that the greater the tachypnea, the more apt is slowing to result from oxygen therapy. The experimental work of Binger, Brow, and Branch is of interest in this connection. When multiple emboli of the pulmonary capillaries and arterioles are produced in dogs by the intravenous injection of suspensions of potato starch, rapid shallow breathing occurs, associated with arterial anoxemia. The inhalation of oxygen elevates the arterial oxygen saturation, but causes only a slight reduction in respiratory rate. The pathologic picture is that of wide-spread pulmonary congestion and edema. This recalls the findings of Barach and Woodwell, who observed that the dyspnea of cardiac insufficiency was unrelieved by oxygen inhalation despite the fact that the arterial oxygen saturation was brought to the normal level. In both instances the shallow breathing resulted from the impaired elasticity of the lungs which could not be altered by oxygen inhalation, although the arterial anoxemia itself was removed.

When, however, multiple emboli of the larger branches of the pulmonary artery are produced by the injection of seeds, the resulting anoxemia and rapid breathing are both removed by the inhalation of oxygen. Section of the lungs shows large areas of pulmonary circulation cut off thus decreasing the vascular diffusion area and increasing the rate of flow through the functioning capillaries. Under these circumstances the hypothesis is advanced that the thickly crowded corpuscles pass through the capillaries so rapidly that they fail to take up their normal load of oxygen. In the above case of pulmonary embolus the relief of dyspnea was striking, the arterial anoxemia was much diminished, and the respiratory rate was considerably slowed by the inhalation of oxygen. In the dog experiments the tachypnea was removed by the inhalation of oxygen, whereas in the human subject it was lessened. In pneumonia the vascular diffusion area is also much diminished, but there are additional influences such as impaired elasticity due to consolidation and pulmonary edema which are added factors in the dyspnea and the arterial anoxemia. The inhalation of oxygen-rich mixtures removes arterial anoxemia dependent on pulmonary edema and a decreased vascular diffusion area, but it has no influence on a heavy inelastic lung. For this reason perhaps the dyspnea in pneumonia is usually only slightly relieved, although the arterial oxygen saturation may be brought almost to the normal level.

SUMMARY

The physiologic basis of the therapeutic use of oxygen is reviewed in the light of modern investigation. Emphasis is placed on the employment of effective methods. The clinical histories of 3 cases of pneumonia and 1 case of postoperative pulmonary embolus are cited. The favorable results that follow adequate oxygen therapy in these conditions are discussed.

CLINIC OF DR ARTHUR F KRAETZER

BELLEVUE HOSPITAL

THE NERVOUS, RUN-DOWN PATIENT

THE problem of the so-called nervous, run-down patient is in a state as neglected as anything in medicine. This type of patient is ubiquitous. If statistics were available, they would probably show him outnumbering sufferers from syphilis and tuberculosis combined. If an ideal efficiency expert could measure in tangible terms the aggregate physical, intellectual, emotional, and creative loss referable to this type of individual—to say nothing of the grouches and futilities occasioned in relatives and friends—the result would doubtless be appalling. And yet he has been given no methodical study. The reason is simple. He is not spectacular. He is merely a nuisance. The routine, so-called tonic, treatment, generally strychnin, is merely a whip to a tired horse. It fails miserably, and yet, despite its pathetic ineffectuality, is prescribed for the next sufferer with a naïve lack of critical insight that is pathetic. The last step is to order a trip to Europe or Florida, a proceeding which a cynical Freudian would interpret, only too accurately, as an attempt to get rid of the poor devil.

For some curious reason the "interesting case" is some exotic rarity or some last stage proposition, both of which examples of disease generally diagnose themselves. The writer once made a tremendous hit on the wards by diagnosing syphilis in a case of juxta-articular nodules. By some curious accident he had seen a similar case two years before. When the Wassermann was reported four plus it was hailed as a marvelous clinical grand-stand play and the applause of the resident intern was flattering in the extreme. But when, in a puzzling case of chronic

cough, he discovered and cured a banal congested lingual tonsil, the lack of appreciation, from the standpoint of his personal vanity, was painful to a degree. Intellectually, there is something almost vulgar, almost pertaining to the parvenu, in this attitude.

The place for detailed clinical study, far more than the ward, is the out-patient department. The ward is not to be neglected, but the dispensary is the field for far more intensive study than has been granted it. It is here that the nervous, run-down

NAME	RES.	TEL.	DATE
AGE	REFERRED BY	BUS. ADDR.	TEL.
FAMILY HISTORY			
CHILDHOOD DISEASES			
PAST HISTORY (Tuberculosis, syphilis, gonorrhea)			
GASTRO-INTESTINAL Appetite		Bowels	Calculus
Dyspepsia	Nausea	Flatulence	Mucosa
Gas	1 Pain		
RESPIRATORY Head-cold		Cough	Expectoration
Hemoptysis			
CIRCULATORY Dyspnea		Orthopnea	Edema of ankles
P. fixation	Cad. extension		
GENITOURINARY Nocturia		Frequency	Diff. in micturition
Menses Onset	1 Premature	1 Duration	Amount
Cycle	1 Pain	1 Last period	
Associated symptoms			
Menopausal symptoms		Post-menopausal bleeding	Childbearing
Laborious	1 Pregnancy	Macromegaly	Childbearing
SKIN Eruptions		P. areas	1 Swellings
GENERAL Endurance		1 Weakness	Reaction to temperature
Night-sweats	Tobacco	Alcohol	Sugar
Tea, Coffee	Average breakfast	Lunch	1 Diet
Drowsiness	Rapid eating	Headache	
PRESENT COMPLAINT			

Fig. 89

patient comes, and it is here that a method, a real method, must be developed and utilized for his care. It is incredible that in this day the so-called diagnosis of neurasthenia should be made in this type of case. Methodical study shows that he is intensely interesting, that he is just as deserving of the admirable routine of the ward service, that he shows pathology that can be demonstrated with the accuracy of the postmortem room, and that he brings the clinician with a jarring thud against physiologic problems that are years from their solution. It is here

that we can learn to formulate our questions, that first step in the answer of any problem. Why is it, for example that a constipated woman is often temporarily relieved of her constipation during her menstrual period? No one knows, but the interrogation has been formulated in the out-patient department, and by its mere formulation we see at once that it probably strikes deep into the unknown of endocrine physiology.

NAME				DATE			
GENERAL APPEARANCE							
Weight	lbs.	Height	inches	Span	inches	Development	inches
Lower extremities		inches		Face			
Scroto-ventral edges				Scroto-ventral fissure			
Neck		Throat				Aortic arch	
Extremities						Hands	
Cervix		Cervix					
MOUTH AND PHARYNX							
Color		crested		tongue		Tongue protruded	
Past				atrophy		dented edges	
Teeth				Gum			
Frenum				Tonsils			
Base of tongue, lingual tonsil						varices	
mouth strictly		Larynx					
Alveoli		Anterior nares					
Nasal mucosa							
Septum							
Turbinate							
Sinus, maxillary							
Transillumination							
EYES							
Lids				Cornea			
Tarsal conjunctiva				Bulbar conjunctiva			
Pupil		Right		Left		Enophthalmos	
Reaction to light				Nystagmus		Crawling	
Accommodation				Pupils		Nictitating	
Conjunctival reflex				Conjunctiva		Dilatation	
Iris				Hernia		Miosis	
Eye grounds							
EARS							
Tact		External meatus					
Tympanic membrane							
HEART							
Point of maximal impulse is		intercostal space,		cm from midclavicular line			
Right		Left		Force		Rate, irreg.	
1 C.S.		Impulse				radial	
2		Rheumatic					
3		Tricuspid					
4		Mitral					
5							
6		Mild first sound					
7		Palpable second		Aortic second			
Corder above as in diagram							

Fig 90

The method used in studying the type of patient under discussion is essentially the same as that used for the ward patient. In addition, however, certain other points are to be considered. The study includes the following

1 History Not the mere description of the presenting symptom, but the following of a set form that takes up all phases of the

patient's past and present pathologic physiology. The presenting symptom is to be taken up only after the form has been filled in.

2 Physical examination. This is to be complete in the most rigid sense of the word. It is to include not merely the standard investigation of the viscera, but a description and listing of those findings in the skin and physical habitus, which, while not yet

NAME		DATE	
VASCULAR Blood Pressure			
Radial arteries		R. S. pulse	
Episternal pulsation		Renal arteries	
Veins of neck		Carotid pulse	
		at lower extremities	
LUNGS Chest			
T. hrs.		Cost. angle	
Expansion		Lungs & signs	
Extremities at base			
ABDOMEN			
LYMPHATIC Nodes: Cervical			
Axillary		Enterohepatic	
Inguinal			
SKIN Color			
Livid		Moisture	
Anorexia in		Sc. to	
Eye-brows		Bred	
Hair: Thoracic		Abdominal	
Pubic		Escrotal	
Nails: Curved		Bred	
Rashes		Lesions	
White Spots			
MUSCULAR Development			
Tenderness		Atrophy	
Cephalic			
BONES AND JOINTS			
NEUROLOGICAL			
Right	Left	Right	Left
Biceps reflex		Contraction reflex	
Triceps "		Pharyngeal	
Opisthorus		Abdominal	
Achilles		Cervicogenic	
Reflex		Ankle clonus	
Romberg	Ataxia		
GENITOURINARY			

Fig 91

on an established diagnostic basis, except in endocrine advertisements, are becoming more and more suspected of furnishing clues to the patient's constitution.

3 Laboratory

(a) Urinalysis

(b) Blood-count, hemoglobin, red, white, and differential

(c) Wassermann

The function of the urinalysis is obvious enough. The blood-count will bring to light the common chlorotic type of anemia, or it may even reveal the rarer types of blood disease. However, it must be clearly understood that anemia is not a diagnosis. It is merely a physical sign. The real diagnosis is the cause of the anemia. An eosinophilia may be the sole clue to an absolutely unsuspected infestation with intestinal parasites.

URINALYSIS		BLOOD PICTURE	
NAME	.	NAME	DATE
DATE			
VOLUME		R B C	
APPEARANCE		HEMOGLOBIN	
COLOR		COLOR INDEX	
REACTION		W B C	
SPEC GRAV		POLY	
ALBUMIN		LYMPH	
SUGAR		LARGE MONO	
ACETONE		EOSIN	
DIACETIC		TRANSITIONAL	
INDICAN		BASOPHIL	
BILE PIGMENT		ABNORMALITIES	
MICROSCOPICAL		<u>WASSERMANN</u>	
CASTS			
CYLINDROIDS			
MUCUS			
CRYSTALS			
W B C.			
R B C			
AMORPHOUS			
BACTERIA			
EPITHELIA			

PROVISIONAL DIAGNOSIS

Fig 92

The Wassermann as a routine is of more importance than the urinalysis. Time and again the only manifestation of syphilis is the picture of so-called neurasthenia. A case of nasal catarrh which had been treated with sprays for years showed a four plus Wassermann and cleared up with a few injections of salvarsan. To ignore the possibility of syphilis merely because the patient shouldn't have syphilis, is loose beyond words. Syphilis is a function of the *Spirochaeta pallida*, not of a patient's social

position In the same way, tuberculosis must invariably be given due consideration in the investigation of the nervous, run-down patient Time and again the very early case, than which nothing is more gratifying to diagnose, is labeled neurasthenia or dyspepsia, and dismissed with a prescription for nux or rhubarb and soda There often seems to be an actual ostrich attitude towards this disease, an attitude based on a lack of courage to face an unpleasant possibility and on an inability to diagnose the condition

The form of the above investigation is shown on other pages It represents a starting-point for special study, although frequently it will be sufficient in itself. If the history and physical findings are suggestive of tuberculosis, then x-ray and sputum examinations can be made If the patient is shown to have syphilis and intestinal toxemia, it is not sufficient to give the standard course of antiluetic drugs The patient has not merely got syphilis He has syphilis plus intestinal toxemia If the latter condition is intelligently treated, the patient will endure his specific treatment much more gracefully This is not only shown clinically, but, inasmuch as the colon is the organ of excretion for poisonous metals, is what is to be expected Furthermore, there is considerable suspicion that foci of infection may, in syphilis, play two very evil rôles, first, in causing Wassermann "fastness," second, in creating a locus minoris resistentiæ in some vital structure and thus rendering it secondarily a prey to the spirochete This is a point well to be considered in opposition to the theory of specific tropism on the part of the spirochete

Assuming that the graver conditions, such as tuberculosis, syphilis, diabetes, etc., have been with reasonable probability ruled out, the following are the common things brought to light in the so-called nervous, run-down patient, and playing the chief rôles in the genesis of his ill health

- 1 Intestinal toxemia
- 2 Focal infection
- 3 Fibrositis
- 4 Constitutional faults of more or less vague nature

There is a curious tradition about intestinal toxemia that it is always due to the putrefaction of animal food, and that it cannot exist if there is a regular daily bowel movement. As a result, the standard treatment is the ordering of a vegetarian diet and the use of cathartics. Despite the repeated failure of this method, it is still persisted in. If one wants to be very scientific he prescribes the *acidophilus bacillus*. The writer, himself, has had indifferent success with this organism. To treat intestinal toxemia it is first necessary to know what it is or, at least, to describe it as a nosologic entity. Exactly what intestinal toxemia is we do not, in the present state of information, know. The supposititious toxins arising in the intestinal tract have not been isolated. It is, therefore, impossible to construct an exact scientific definition of the condition. It is, however, possible to describe it in terms of a clear-cut nosologic entity. For two years the writer treated his cases with vegetarianism and colon irrigations, with mediocre success. He noted that the cases showed two findings, a tongue indentated along the edges by the teeth, and an almost universal absence of indican in the urine. This latter was a discrepancy at variance with the assumption of a protein putrefaction. It was dismissed on the grounds that if indican were not present, at any rate, it ought to be. He then became acquainted with some work of the late Dr. Johnston, of the Dermatology Department of Cornell, who described a vaguely defined white streak on the vermilion of the upper lip, and running through tests on several thousand individuals, found that it was associated with a fermentative stool. The writer then noted that this white line was invariably associated with the slightly edematous tongue, indentated along its lateral margins by the teeth, and *almost* invariably with an absence of indican. Of the exceptions to this last finding, more will be spoken later. It thus became possible to construct a well-defined trune entity, comprising, (1) the white line on the labial vermilion, (2) the indentated tongue, (3) absence of indicanuria, an entity which could be simply and accurately determined objectively, without reference to any of the subjective complaints or symptoms of the patient. These last, of

course, like most subjective findings, are manifold and ambiguous. They are cramp-like pains in the hypogastrium, burning pain in the epigastrium, vague abdominal pains of indefinite location, flatulency, sour eructations, foul-smelling stools, bromidrosis, acne, folliculitis, muscular aches often worse in damp weather, general malaise, nervousness, insomnia, easy fatigue, and "lack of pep." Most of the patients have regular bowel movements, and for this reason constipation, though present in the form of retained scybala and mucus, is not suspected until the retained products are brought to light by enema.

It is appropriate to insert a word here about the characteristics of the patient's stools. Scybala, hard lumps with a corrugated, raisin-like surface, are always present. Not infrequently they may be felt during digital rectal examination. They are probably caused by the action of a spastic sigmoid, and the latter can be easily palpated deep in the left lower quadrant, as a hard rounded cord. The writer has observed these scybala at operation, filling the transverse colon. It may be that in these cases they are formed *in situ* by a spastic colon, or possibly they have been formed lower down in the sigmoid and then swept back by retroperistalsis. At any rate they represent a semi-permanent accumulation, and are paid out in small doles, the colon never being entirely free from them. Cathartics and the ordinary enema seem to be very ineffectual in removing them, but the ichthyol enema seems to have peculiar success in so doing. Cathartics increase the spasticity which causes the scybala and aggravate the colitis which causes the mucous formation.

At times the normally passed stool is of the ordinary formed character, with numerous scybala incorporated in it, the whole giving an appearance suggestive of the conglomerate rock of the geologist. This can only mean two distinct editions of fecal formation, one recent, the other comparatively ancient. It means that in spite of daily movements, constipation is always present. In these cases the ichthyol enema first removes recent fecal matter and old scybala. At the very end of the evacuation there often appears a dark, soft, amorphous mass of foul material, with a glistening, flaky, mucid appearance. Its late appearance.

its excessive foulness, and the evidence of low-grade colitis, in the shape of mucus, probably mean that it is very old material from high up in the colon. Coincident with its passage the patient experiences a sense of sudden relief.

In the light of Johnston's findings, the absence of indicanuria is easily understood, and the treatment must logically be a complete reversal of the traditional one. The writer uses in these cases

1. A low carbohydrate diet, absolute avoidance of sugar, potatoes, cereals, bananas, and all flour-containing foods except one slice of bread with each meal. Meat, fish, eggs, milk, and green vegetables are freely taken, with a moderate allowance of fruit.

2. An enema consisting of 2 quarts of warm water plus 2 drams of Merck's ichthyol, given with slight force from a pressure bottle, two or three times a week, with a short rectal tube, the patient lying on the left side and then turning on the right. The ichthyol enema, given in this manner, is more efficacious than the colon irrigation, is easier on the patient, and is infinitely simpler to administer. It is absurd to say that it cannot reach the cecum, for the ordinary barium enema certainly does.

3. A tablespoonful of Pillsbury's bran stirred up in a glass of water night and morning.

4. At least 2 quarts of water daily.

5. A tablespoonful of mineral oil night and morning.

6. Strict avoidance of cathartics.

7. Absolute regularity in going to stool.

The above treatment is extraordinarily simple and extraordinarily effective. Symptoms clear up with a marked promptitude and are replaced by a sense of well-being that the patient has not known for years. As the patient's local and general condition improves the enemas can be tapered off and carbohydrate cautiously increased. In the writer's hands this method has not failed to cure a single case of constipation. A striking thing is the dearth of cases of indicanuria in office and clinic. Practically every case of intestinal toxemia presents the above triad of findings and responds to the low carbohydrate diet.

This is probably referable to the extraordinary consumption of sugar in this country, which, one hundred years ago, was 5 pounds per head per year, and is today 102 pounds per head per year. To refer to the exceptions mentioned in which indicanuria occurred with the white labial line and the indented tongue, 3 cases who presented themselves to the writer with the last two findings were told to go on a strict low carbohydrate diet. After they had left the office their urine was examined and found to contain large amounts of indican. It was felt that they had been given absolutely wrong advice, but when they returned a week later, their condition was improved, and, remarkable to state, the indican had disappeared, and this in spite of the fact that they were eating a relatively and absolutely greater amount of indican-producing foods. The only plausible explanation of this fact is the hypothesis that a mucosa damaged by fermentative processes may no longer be impervious to substances like indican. The fact itself certainly suggests that the generally accepted belief that fermentative and putrefactive flora are mutually incompatible is not necessarily true. One other curious urinary change occurred in two patients. Two young men with albuminuria, presumably the albuminuria of young adults without kidney lesion, while under treatment for carbohydrate fermentation, showed over a period of two to three weeks a progressive, and finally a complete, clearing up of the albumin. The significance of this can only be speculated on. It might be well worth following up.

The subject of focal infection is more clearly understood, but the rôle of such infection in the run-down patient is not sufficiently appreciated. The usual infectious foci in this type of patient are located in the mouth, nasopharynx, and accessory sinuses. They comprise

- 1 Teeth

- (a) Periapical abscess

- (b) Pyorrhea

- 2 Tonsils

- 3 Sinuses

The subject of teeth is trite enough, but its very banality

results in the frequent overlooking of the source of intoxication. A single, painless, peri-apical abscess may play havoc with the individual's health, and in the run-down case every devitalized tooth, even those roentgenologically negative, must be looked on with suspicion and extracted if all other lines of investigation prove negative. A man on one of the writer's wards in one of the Army Camps ran a low-grade septic temperature for two weeks, with absolutely no other symptom. Every conceivable line of investigation was followed, blood-culture, sputum examination, chest x-ray, urine culture, sinus x-ray, search for malarial organisms, all were negative. A single devitalized tooth in an otherwise faultless mouth was removed, and prompt recovery followed. In the indescribably foul mouths that so frequently occur, there is but one thing to do if the patient's health is to be restored, ruthless extraction. *Ubi pus, ibi evacue*.

There is a wide-spread misunderstanding of what constitutes a diseased tonsil. Unfortunately, it is frequently felt that to be diseased a tonsil must be either enlarged or the occasion of sore throat. Time and again a physician looks in a mouth and pronounces the tonsils all right, when, as a matter of fact, he hasn't even seen them, the reason being that, as a result of chronic infection and scar-tissue formation, they are atrophied and shrunken out of sight. Although it is impossible to diagnose healthy tonsils without seeing them, it is possible thus to diagnose diseased ones. If, on the anterior pillar, there is a sharply defined area of dull red congestion, then there is certainly pus in the underlying tonsil. If the anterior pillar is retracted and the tonsil brought into view, steady pressure on the latter with a tongue-blade will express the pus. Such tonsils are sources of impairment to the health and must come out. The symptoms caused by chronically diseased tonsils are, like those caused by intestinal toxemia, manifold. And here a universal principle must be emphasized, that, although the sources of toxic absorption are few in number, their manifestations in the form of symptoms are unlimited in number and variety. Too often these *symptoms* are dignified with the names of *diseases*, and the paralyzing effect of nomenclature prevents search for the true

and more deeply lying cause. A case of purpura, recurring in frequent crops, was referred to the writer's medical service in the New York Skin and Cancer Hospital. The patient, a young adult woman, also complained of constipation, easy fatigue, and severe dysmenorrhea (a symptom, not a disease). She showed (1) A physique tending toward that of the Frohlich type of dystrophia adiposogenitalis, (2) the signs mentioned above as associated with carbohydrate fermentation, (3) chronic atrophic tonsillitis, and (4) two painless abscessed teeth. The routine treatment for carbohydrate fermentation, plus the administration of pituitary gland and corpus luteum, was followed by prompt cure of the constipation and dysmenorrhea, and lessening of the fatigue. She was then considered in good enough shape to undergo tonsillectomy. This was done, with resulting increase in sense of well-being, but with no effect on the purpura. After several weeks the diseased teeth were extracted, and the purpura cleared up promptly. Incidentally, though previously sterile over a considerable period of married life, she became pregnant. To ascribe this to a specific effect of the pituitary feeding is to subject the principle of *post hoc, propter hoc*, to too severe a strain, although, inasmuch as the patient was of a low-grade Frohlich type, a class notoriously unfruitful, the temptation to do so is strong. At any rate, the fact is worth recording as a single isolated incident. Other examples of long-continued sterility becoming pregnant following pituitary feeding have been noted. One deduction, however, can be drawn from the consideration of a case such as the above, that, to paraphrase Allbutt, diseases, so-called, are not entities as such, but are the equivocal expressions of the interplay of manifold and complex variables.

With regard to diseased tonsils the following symptoms have, under the writer's observation, cleared up promptly after tonsillectomy—sciatica, headache, muscular pains, malnutrition, and asthenia. The headache of chronic tonsillitis is not a fairly well-defined clinical picture, as are some of the sinus headaches. It is probably an expression of an underlying fibrositis, as indeed are the sciatic and muscular pains that are relieved by tonsillectomy.

Transillumination of the sinuses should be a matter of the strictest routine. It takes about one minute. It may reveal the main source of a patient's ill health. Time and again chronic empyema of the antrum may be silent, giving no local symptoms whatever, in the shape of pain or discharge, but nevertheless being responsible for an intolerable condition of general wretchedness. As regards chronic infection of the ethmoid and sphenoid, diagnosis is the function of the expert nose and throat specialist, plus the aid of the x-ray. The internist, however, should be thoroughly conversant with the headaches and eye disorders caused by these conditions, and described by Sluder in what is one of the most valuable clinical contributions of all time.

The subject of fibrositis could well monopolize an entire paper itself. Though a ubiquitous condition, and responsible for a goodly proportion of all human aches and pains, it is given little or no attention in the medical schools. In our own language the English clinicians, particularly Llewellyn, Jones, and Williams, are the only writers giving it adequate attention. Apropos of Edinger's statement, that it is probably the most frequent cause of headache, a statement that is indubitably true, a prominent American diagnostician remarks, "I have, I regret to say, no cases in my own experience that exemplify this disease."

Fibrositis, when mentioned, is generally referred to as chronic muscular rheumatism, although it is not muscular, nor is it rheumatism. It never causes endocarditis. Fibrositis is a phenomenon caused by some distant source of intoxication, expressing itself in pain and tenderness in connective tissue generally, but more particularly in the tendinous insertions of the muscles, and frequently showing an unquestionable relationship to changes in the weather. It has recently become fashionable to turn up one's intellectual nose at the idea of damp cold being related to the exaggeration of aches and pains. That such is the case, however, is an insuperable fact and there are unlimited instances which verify this. The true cause, however, lies deeper.

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be acute and atrociously severe. Given the appropriate location and severity, it may simulate renal colic, gall-bladder colic, and pleurisy, and it has even been mistaken for angina pectoris. A patient was referred to the writer with a diagnosis of angina. He complained of precordial pain radiating down the left arm, and brought on by exertion. A little consideration showed several discrepancies. The pain was precordial, not substernal, which is characteristic of angina. It radiated down the outer, not the inner, side of the arm. It was not associated with stenocardia, nor with the well-known and sinister sense of dread. Further questioning brought out the fact that it was only a particular kind of exertion that occasioned the pain, pushing a lawnmower and swimming, *i. e.*, the exertions putting stress on the muscles of the shoulder-girdle. Climbing stairs, the worst form of strain for a diseased circulatory system, never brought on the pain. Atrophic, pus-containing tonsils were removed, a silent antrum containing indescribably foul pus was washed out, and a month later the imitation angina had cleared up. A condition which is as efficient and versatile a masquerader as this is certainly deserving of more attention from the internist than has been afforded it.

The pathology of fibrositis consists of round-cell infiltration and exudation among the fibers of connective tissue. It may be acute and exudative, later chronic and productive. The sites of election are the insertions of the occipital and gluteal muscles, the aponeurosis of the scalp, the trapezi, the intercostals, the dorsal muscles, the lumbar muscles, and the ligaments of the spine and sacro-iliac joints. Lumbago is simply fibrositis of the lumbar region. Sciatica is often due to fibrositis somewhere along the course of the sciatic nerve, not infrequently in the periarticular structures of the sacro-iliac joints. Physical examination in the acute cases shows marked tenderness and spasm in the affected region. If in the intercostal muscles, it may cause a unilateral diminution of chest movement and breath sounds and thus give rise to the suspicion of pleurisy or pneumonia. The old term of pleurodynia was the tag attached to many of these cases. More chronic cases, on deep

palpation, show gritty, sand-like, tender nodules in the involved structures. These are the indurations in the cases of so-called indurative headache, unquestionably the most frequent type of this symptom. The cause of the condition lies in some distant focus of toxic absorption, particularly tonsils, intestinal tract, teeth, and sinuses. Under the writer's observation cases of occipital headache, stiff and sore neck, pain in the pectoral and deltoid muscles, have cleared up promptly following tonsillectomy. Similar results have followed the extraction of diseased teeth. The relations of the intestinal tract are particularly interesting. One patient with chronic pain and stiffness of the back of the neck and the interphalangeal joints experienced complete relief after a few weeks of a low carbohydrate diet. Nothing else was done. In this case an even moderate indulgence in candy is followed by a return of the symptoms. Another patient gets marked relief for his occasional mild exacerbations by taking calomel or an ichthyol enema, and the relief is exceedingly prompt. This is of particular interest in the light of the recent discussion regarding the relief from malaise following bowel movement. It is argued that the *promptness* of the subjective improvement must mean that it comes from relief of mechanical distention and pressure, and that a single evacuation cannot to any degree diminish a toxic saturation, in other words, that you cannot cure a drunkard by taking his bottle away from him. Notwithstanding all this, the relief that this case of fibrositis gets from calomel or enema can only come from a diminution of his toxemia, for it is inconceivable that pains in the back of the neck, the shoulders, and the finger-joints could be dependent on mechanical pressure in the lower bowel. In the writer's opinion there is no doubt that the prompt effect of colonic cleansing is due not to its mechanically but to its chemically, purifying effect. To return from this digression, it is highly probable that in some cases of fibrositis carbohydrate fermentation represents the original source of toxic absorption.

The cases of fibrositis, besides their local aches, also frequently suffer from general malaise, nervousness, and easy fatigue, and well they may, because they are the subjects of a wide-spread,

low-grade intoxication This is the type, *par excellence*, which is so frequently and so unjustly dubbed neurotic, and the sensitive spine, far from being considered in its true light as evidence of tangible inflammation, is looked upon as something in the nature of an hysterical stigma

The first and most important part of treatment is to clear up all sources of toxic absorption, particularly the intestinal tract The thing of next importance is massage Next, diathermy and the static spark Local heat is of value, but the internal heat of diathermy is more effective Counterrrritation will, once in a while, be effective, and the mustard plaster is by no means obsolete, particularly in the acute cases As a rule, however, it is disappointing The other methods are just as easy to use, and much more efficacious Vaccines will help at times, but in the presence of infectious foci register more failures than successes In the presence of diseased tonsils, teeth, or sinuses surgery is imperative Half-way measures are not good medicine Furthermore, it must never be forgotten that fibrositis, in spite of its attractive Latin name, is not a disease *per se*, but, notwithstanding the urgency of the discomfort it occasions, has a merely epiphenomenal significance, and is to be looked on as the expression of a really sinister underlying intoxication

To treat the last heading under which the nervous, run-down patient must be discussed, namely, constitutional faults of more or less vague nature, requires considerable courage It leads us into the little understood field of autonomic and sympathetic physiology, wherein lies the Promised Land of internal medicine It also leads into the domain of endocrinology, where so much is known that is not so Empiricism, however, though a dull tool, has fashioned many fine and permanent structures, and in this field will some day lead to something worth while As a working hypothesis we can posit the following The autonomic, or vagal system is stimulative to the vegetative functions, secretion, peristalsis, etc The sympathetic is inhibitory Perfect function with a parallel sense of well-being involves a dynamic balance between these systems The vagus is possibly stimu-

lated by the thyroid, the sympathetic by the adrenal. The condition known as vagotonia, which embraces an enormous number of nervous run-down people, may be due to essentially too much vagus, but seems due rather to too little sympathetic, with a secondary, relative predominance of the vagus. And this, in turn, goes back to a probably deficient adrenal. The arch type of this condition is the so-called status lymphaticus, which shows the following points:

1 Deficient beard, thoracic and abdominal hair. Feminine distribution of pubic hair (deficient adrenal).

2 Tendency toward feminine configuration of body, rounded rather than angular outline, narrow waist, large hips and buttocks, curving thighs, genu valgum.

3 Soft, transparent skin. Deficient pigment (deficient adrenal).

4 Actual deficiency of the adrenal cortex.

5 Persistent thymus (antagonistic to the adrenals and gonads).

6 Deficiency of arterial structure, narrow aorta, thin-walled cerebral vessels, an occasional cause of apoplexy in the young (possible result of faulty trophic action of deficient sympathetic).

7 Respiratory arrhythmia (vagotonia).

8 Hyperchlorhydria (vagotonia).

9 Pylorospasm (vagotonia).

10 Spastic sigmoid (vagotonia) causing constipation and the formation of scybala.

11 Easy fatigue. Easy fainting (poor sympathetic control).

12 Acro-asphyxia, or cyanosis of the hands (poor sympathetic control).

13 Livido, or annular cyanosis of the skin (poor sympathetic control).

14 Liability to anaphylactic phenomena, such as asthma, hay-fever, urticaria, toxic dermatitis (poor sympathetic control).

15 Low resistance to infection. An enormous number of cases of status lymphaticus is found among tuberculous subjects.

16 Sudden death from disproportionately insignificant

causes, such as intravenous injection of serum, etc (mechanism not understood)

17 Sergent's line (supposedly a sign of deficient adrenal)

18 Low-grade eosinophilia

19 Physical and emotional instability Insufficient adaptability to environmental stress At times an actual criminal tendency This, perhaps, like the phenomena of status lymphaticus in general, may possibly be referred to an atavistic trend

20 Asthenic habitus, enteroptotic configuration, acute costal angle, protuberant lower abdomen, long narrow chest

There is an infinity of variants on the above scheme Atropin, by depressing the vagus, tends to restore the imbalance between the bulbosacral and sympathetic systems, and will often abolish some symptoms, particularly hyperchlorhydria and its associated dyspepsia Theoretically, adreno-nucleo-protein and adrenal residue should have a corresponding effect The results, however, are disappointing The writer has seen 2 cases of toxic dermatitis show what was apparently a clear-cut response to adrenal residue The effect of adrenalin on an asthmatic attack is well known

As it is impossible to generalize accurately in this difficult and little-known field, it may be better to describe individual cases A man of thirty-four complained of nervousness, irritability, fatigue, and severe burning epigastric pain without definite relation to meals, but invariably worse at night His bowels were regular Gastric series showed no abnormality, and a test-meal showed a marked excess of acid The diagnosis of hyperchlorhydria was made Again we see a symptom or, rather, a physical sign mistaken for a disease He was treated for six weeks without result The treatment included the administration of olive oil and adrenal residue Examination showed a slim, small-boned, asthenic man, with acute costal angle, slightly protuberant lower abdomen, marked thinning of the outer half of the eye-brows, scanty thoracic hair, feminine pubic hair, white streak on the vermilion of the upper lip, a coated tongue indented along the edges, tenderness over the epigastrium and sigmoid, a slow pulse and a marked respiratory

arrhythmia Sargent's line was not present The following provisional diagnosis was made

- 1 Status lymphaticus
- 2 Vagotonia
- 3 Carbohydrate fermentation.
- 4 Lax abdominal musculature
- 5 Hypothyroidism(?)

He was put on a strict low carbohydrate diet, mineral oil, ichthyol enemata, and atropin sulphate, 1/200 grain three times a day. In two days he was completely free from pain, and from then on his strength and sense of well-being steadily increased. Later an abdominal belt was ordered, but this seemed to be superfluous. As not infrequently happens, the ichthyol enemata were followed by temporary constipation. This, however, cleared up promptly, as is invariably the case.

It is well to note in this case certain discrepancies from the hypothesis mentioned at the start of the discussion of these vague constitutional faults. Thinning of the other half of the eyebrows is probably a fairly accurate sign of hypothyroidism, or perhaps it would be more correct to say that many patients with this sign are helped by thyroid feeding. At the same time many of these individuals are definitely vagotonic. It is hard to see how thyroid can be a specific vague stimulant and at the same time, by its administration, cause improvement in vagotonic conditions. Thyroid was not given to the above case, but has been helpful in similar patients. The hypothetical dynamic antagonism between the vagus and the sympathetic is probably correct, but it is highly doubtful if there is a corresponding antagonism, at least as clear-cut an antagonism, between the thyroid and the adrenal. As has been mentioned, the above patient was treated with adrenal residue without success.

Many of these cases show a lax, protuberant, lower abdomen, and are diagnosed as enteroptosis. It is undeniable that many of them get great relief from the abdominal belt, but that this results primarily from replacing a "dropped stomach" is highly questionable. It is more probable that the laxity of the ab-

dominal muscles, plus concomitant anatomic variations, such as a long narrow thorax with acute costal angle, causes the viscera to exert a constant drag on the abdominal sympathetic, the resulting symptoms being fatigue phenomena. Low blood-pressure is a frequent finding in these cases, and there have been recently reported some very interesting examples of low blood-pressure showing a rise of 10 to 20 mm after putting on a well-fitting abdominal binder. This may result from relieving the drag on the sympathetic plexus, but it is more probable that the resulting application of compression to the lax abdominal, vascular lakes is the real factor. It is certainly a fact that many cases of postinfluenzal asthenia are considerably helped by temporary abdominal support.

One other therapeutic agent of great value in the nervous, run-down patient is the ultraviolet lamp. A few general exposures to the point of erythema and tanning cause a marked increase in the sense of energy and well-being. This is not surprising, for if the ultraviolet lamp can stimulate resistance to the point of overcoming bone tuberculosis, it should also be capable of stimulating resistance to the point of overcoming lesser conditions.

This paper ends where it began, with a plea for the more thorough and conscientious study of an innumerable group of individuals, whose sufferings are very real and are not capricious, but are based on definite physical defects capable of recognition and correction.

CONTRIBUTION BY DR I W HELD

CLINIC OF BETH ISRAEL HOSPITAL

ICTERUS AND ITS DIFFERENTIAL DIAGNOSIS

ICTERUS is an objective symptom which is most easily recognized, but which offers great difficulty in diagnostic interpretation. Icterus in every case is the result of the accumulation of bile pigments (mainly bilirubin and, to a very small extent, biliverdin) in the tissues. This is true even in those cases in which no bile pigment is present in the urine, acholuric icterus, where, instead of bilirubin, there is urobilin in the urine.

According to our present state of knowledge, icterus can be divided into (1) the obstructive type with an obstruction to the flow of bile in the ducts, (2) the hepatic type which is due to functional or organic changes in the liver cells of either infectious or toxic origin, and (3) the hemolytic type. Whereas, it is agreed that the disturbed function in the liver cells is responsible for the icterus in the first two types, the hemolytic form has given rise to considerable discussion regarding the participation of the liver cells in the production of icterus.

For a proper understanding of the pathogenesis of icterus it is essential to review the physiology of the most important constituents of bile, namely, bilirubin, urobilin, and bile acids.

BILIRUBIN

Up to the time of the discovery of Virchow¹ that hematoidin can be formed from hemoglobin in blood extravasations and the chemical proof by Jaffé² that hematoidin is identical with bilirubin, it was considered an established fact that bilirubin is formed in the liver cells. The work of Virchow and Jaffé laid the foundation for the theory of hematogenous icterus, although

Morgagni clinically described the same form under the name of suppression icterus

The discoveries of Virchow and Jaffé marked a new era of research in the problem of icterus and confirmed the theory that bilirubin can be formed outside the liver cells. The increase of bilirubin and bile in the blood after transfusion or that due to hemolytic poisons, experimental bilirubinemia and icterus in animals poisoned with toluylendiamin or arseniuretted hydrogen, or that occurring in paroxysmal hemoglobinuria, is strong evidence that bilirubin is formed in other organs and that it is brought to the liver to be converted into bile. Lowitt,³ who experimented on frogs in whom the phagocytic action of the endothelial cells in the liver (Kupffer stellate cells) are most easily studied, found that there is a large deposit of iron and bilirubin in these cells in hemolysis. This laid the foundation for the work of McNee,⁴ a pupil of Aschoff,⁵ and to the theory of Aschoff and McNee that bilirubin is formed in the reticulo-endothelial system. This system, as is well known, consists of the Kupffer stellate cells in the liver, the sinuses in the spleen, and the hemolymph glands. The theory of anhepatogenous formation of bilirubin gained such ground that even icterus without the presence of the liver was considered possible.

In 1886 the work of Minkowski and Naunyn⁶ swung the pendulum in the opposite direction when they showed that if the liver is extirpated from geese and ducks and arseniuretted hydrogen is injected intravenously in order to produce hemolysis, not only is it impossible to produce icterus, but bilirubinemia cannot be brought about. Hemolysis brought about in a similar way in control animals caused icterus and bilirubinemia. These experiments were rightfully accepted until McNee in Aschoff's laboratory repeated the experiments of Minkowski and Naunyn, with similar results, but his interpretations were different. McNee concludes that jaundice did not appear, not because the liver cells were removed, but because the Kupffer stellate cells were removed.

McNee's work convinces him that bile pigment is formed in the reticulo-endothelial system exclusively. On the basis of this

theory he states that icterus can arise under the following conditions. First, where bile pigment formed in the cells of the reticulo-endothelial system passes through the polygonal cells to reach the bile capillaries, but is obstructed there and reabsorbed into the blood. Under such conditions the direct H₁zman van den Bergh bilirubin reaction is present. Second where, owing to damage to the liver cells, the bile carried there is unable to enter and the bilirubin passes, therefore, along the hepatic venous radicles into the general circulation. On the other hand, part of the bilirubin might pass through and part be unable to do so. Under these conditions the biphasic bilirubin reaction is present, which is a combination of prompt direct and delayed direct reactions. Third, where in excessive blood destruction too much bilirubin is formed in the reticulo-endothelial system for the liver cells to deal with. In such cases some pigment might pass normally into the bile capillaries, while the excess might pass straight through the hepatic vein into the circulation and bring about jaundice. Under such conditions the indirect bilirubin reaction is present but not the direct. Fourth, where, in addition to the damaged liver cells, there is likewise obstruction in the bile-ducts, as, for example, in cholangitis. McNee's clinical classification of icterus corresponds to the classification already mentioned above, namely, (a) obstructive, (b) hepatic (which he calls infectious or toxic), and (c) hemolytic.²⁶

Independently, Whipple and Hooper⁷ found that the removal of the liver in dogs does not interfere with the formation of bilirubin and even with the production of some degree of icterus. The method employed by Whipple and Hooper was the production of an Eck's fistula, ligation of the hepatic artery, and the establishment of a cephalic and thoracic circulation by ligation of the aorta just below the subclavian artery and, again, at the level of and including the celiac axis, and ligation of the inferior vena cava above the diaphragm and of the mammary vessels in the thorax. They also twisted wire ligaments along the margins of the ribs. Laked red blood-corpuscles were then introduced into the circulation by way of the jugular vein. In spite of the absence of the liver, bilirubin was found in the blood and a defi-

ntely visible tissue icterus was produced Their experiments strengthened the theory of anhepatogenous bilirubin formation

On reviewing the results of his own work F C Mann⁸ of the Mayo Clinic arrives at the following conclusion That following hepatectomy in dogs there is a gradual accumulation of a yellow pigment in the urine, plasma, and fat In animals who survive for twenty-four hours or more there is a definite yellow discoloration of the mucous membranes and the scleræ The urine, plasma, and fat give positive tests for bilirubin after four to six hours and they show definite progressive increase in the bilirubin content as long as the animal survives The Hymann van den Bergh reaction is at first indirect and becomes biphasic about twelve hours after hepatectomy The fact that the bilirubin is not dependent on reabsorption from the gastrointestinal tract or any of the abdominal viscera is shown by its appearance in an approximately similar amount after the removal of the liver and entire abdominal viscera Extravasated blood in the body cavities or tissues is not a factor in these experiments, as bilirubin could be demonstrated in several animals in whom no trace of hemorrhage could be found at necropsy Injections of laked blood-cells in the hepatectomized animals cause marked increase in the bilirubin content of the plasma According to the work of Mann, therefore, bilirubin is formed at a definite rate without the intervention of the liver, spleen, or other abdominal viscera These findings indicate that the liver is not essential for the formation of bilirubin and that it should be considered perhaps mainly as an excretory organ so far as bilirubin is concerned The main support for the anhepatogenous theory of bilirubin formation is, therefore, the work of Whipple and Hooper, and that of Mann

Naunyn, who strongly defends the hepatogenous theory of bilirubin formation, reasons that it is true that in the endothelial cells of the liver, spleen, and hemolymph glands yellow and green crystals are formed which must be considered as bile pigment, but he does not agree that the quantity of these pigments plays an important rôle in the formation of bile Naunyn states that the Kupffer cells take up all material brought there,

including bile pigments through their phagocytic property, but that the pigments are not formed there. Bile casts formed in the bile capillaries are also found in these endothelial cells.

Under physiologic conditions, therefore, the pigment found in these endothelial cells would, according to Naunyn, have no important influence on the formation of bile. Under pathologic conditions, however, the bile pigments formed outside of the liver may play an important rôle in the production of bile. Bile pigments are formed within and outside of the cells in case of excessive destruction of erythrocytes and when there is free hemoglobin in the tissues. The microscopic yellowish-red hematoidin crystals in hemorrhagic effusions, exudates, and cardiac failure cells have been known for a long time. Under pathologic conditions, as for instance, in diseases of the blood, such as pernicious anemia, aplastic anemia, and in hemolytic splenic tumors, the bilirubin in the blood of the splenic vein is actually increased in comparison with that in the crural vein. This was recently again pointed out by Leschke.

Almost indisputable proof of the sole importance of the liver in the formation of bilirubin, supporting the Minkowski-Naunyn work, was furnished by the ingenious experiment of Rich⁹ in Johns Hopkins University and by Retzlaff¹⁰ in the clinic of Kraus. Rich removed the liver from dogs and also repeated the experiments of Whipple and Hooper, using even greater precautions against the establishment of collateral circulation, and he injected the animals with hemoglobin. No bilirubinemia or jaundice was produced. His work does not exclude the possibility of the Kupffer cells being the place where bilirubin is produced.

Retzlaff produced Eck's fistulas in dogs and ligated the hepatic artery and then poisoned them with phenylhydrazin, and he found no bilirubin in the blood.

Experiments performed by F. Rosenthal¹¹ in the clinic of Minkowski, and those by Bieling and Isaac¹² in the clinic of von Noorden tend to cast doubt on the importance of the reticulo-endothelial system in the production of bilirubin. The former blocked the Kupffer stellate cells by injecting the animals intra-

venously with collargol, and he then produced hemolysis. In spite of this blockage bilirubinemia and even icterus were produced. Bieling and Isaac removed the spleen and blocked the Kupffer stellate cells and by the process of hemolysis produced bilirubinemia and icterus, which showed that without the endothelial cells in the sinuses of the spleen, and without the Kupffer stellate cells, jaundice and bilirubin can be produced. This would tend to prove that the only place for the production of bilirubin is in the liver cells. The work of Beckman,¹³ proving that the pigment and hematoidin formed in the extravasations of the blood is different from bilirubin in the bile, is interesting and deserves mention. He injected animals with pigments from extravasated blood and did not succeed in producing bilirubinemia, but if the pigments obtained directly from the bile were injected, bilirubin was produced. Beckman also reasons that, the fact that bilirubinemia, the cause of hemolysis, gives the indirect reaction with the Ehrlich reagent (Hijmann van den Bergh)* would also tend to prove that the respective pigments are not the same. Although the work of the above-named authors is very interesting, it seems to me that they failed to prove that the reticulo-endothelial system is not concerned in the production of bilirubin and that the bile pigment in the extravasation of the blood or that due to hemolysis is essentially different from the bile pigments formed in the liver cells. Against the blockage experiment with collargol it may be said that the Kupffer cells which take up all foreign substance may, in addition to that, also have another physiologic property. The phagocytic storing up of substances such as iron, remnants of red blood-cells, and free

* The Hijmann van den Bergh test for bile pigment in the blood serum depends on the Ehrlich diazo reaction. Freshly prepared mixture of diazotized sulphanilic acid and sodium nitrite is added to the serum. If a pink color appears at once, it is called a direct reaction, if only after the addition of alcohol the color appears, it is known as an indirect reaction. If there is a pink color at once and which deepens on the further addition of alcohol, it is called a biphasic reaction. The direct reaction is present if the jaundice is of hepatogenous origin, so that the bile in the blood serum directly comes from a disease of the liver or ducts. If, on the other hand, the bilirubin in the blood is the result of excessive blood destruction, the indirect reaction results. The bile pigment remains combined with the protein.

hemoglobin is one function of the Kupffer cells and the conversion of hemoglobin into bilirubin is another

The work of Beckman, which is intended to prove that the pigment of the blood extravasations acts differently from that of bile, does not necessarily indicate that these substances are chemically different. It merely shows that the effect of the respective pigments depends more upon their source than upon their chemical character.

From the present state of knowledge, therefore, it seems reasonable to adhere to the teachings of Virchow, Jaffé, Minkowski, Naunyn, Lepehne, and Eppinger, that hematoidin and bilirubin are identical, and that the bile pigments can also be formed outside the liver cells and most likely in the reticulo-endothelial system.

Under physiologic conditions the bilirubin formed outside the liver cells is insignificant. The main formation takes place from the hemoglobin in the liver cells. Under pathologic conditions, as in cases of large hemorrhagic effusion, or in cases of hemolysis in the body either of bacterial or toxic nature, the bilirubin produced outside of the liver cells is of sufficient quantity to be absorbed in the blood and cause an increase in the indirect bilirubin in the blood, thus explaining the origin of hemolytic icterus.

UROBILIN

Urobilin occurs in the form of urobilinogen and is a constant constituent of the bile. It is always found in small traces in the urine and in hemorrhagic exudates. It is increased in many febrile diseases, especially scarlet fever, and in cases where there are parenchymatous changes in the liver cells. There is almost uniform agreement with the work of Fr. Müller that urobilin is formed in the intestines from bile. The quantity of urobilin depends upon the amount of bilirubin that reaches the intestines, there it is reduced by intestinal bacteria into urobilin. In intestinal putrefaction most of the bilirubin in the intestines is converted into urobilin. Part of the urobilin enters the portal vein, whence some of it enters the circulation and some returns to the liver cells. In cases of complete biliary

obstruction no urobilin is found in the urine. Recent work (by Retzlaff, loc cit) likewise serves to prove that an increase of bilirubin in the intestines causes an increase in the urobilin in the urine. He showed that if bile is introduced directly into the intestines through the duodenal tube, there is an increase of urobilin in the urine, and an increase in the indirect bilirubin in the blood. Retzlaff's work tends to show that the bilirubin or bile in the process of absorption in the intestines passes through the intestinal wall and is taken up by the blood. The question arises as to whether the absorption of bile occurs by way of the portal vein into the liver and from there into the lymph stream. After the introduction of large quantities of bile into the intestines, neither the blood of the portal vein nor the lymph of the thoracic duct shows an increase in the direct bilirubin, only the indirect bilirubin being present. If the increase of bile in the intestines causes an increase of indirect bilirubin in the blood, it is reasonable to conclude that the bilirubin normally contained in the blood is due to the absorption of bilirubin from the intestines. If a moderate increase of bile in the intestines can produce bilirubinemia in the healthy subject, it may be assumed that if the intestines are filled with pleochrome bile, that moderate bilirubin will be present in the serum, which would explain the bilirubinemia in hemolytic icterus. It would also explain why the duodenal contents show an increase in bile and a corresponding increase of bilirubin in the blood in hemolytic icterus, in pernicious anemia, aplastic anemia, and in Vaquez-Osler disease, the duodenal contents show a diminution in the amount of bile and the blood shows less bilirubin. According to Retzlaff, therefore, hemolytic icterus could in some cases be explained by the excess of pleochrome bile in the intestines as a result of pathologic hemolysis in the spleen.

Bile acids are formed in the liver from protein and are excreted with the bile into the intestines, where they are partly decomposed and partly reabsorbed. A small part reaches the urine. The bile acids are usually increased in the urine in most cases of icterus with the exception of hemolytic icterus and dur-

ing certain stages of catarrhal icterus Those cases of icterus in which the bile pigments are increased in the urine and bile acids are absent the French term "dissociation icterus "

THE NORMAL SECRETION AND ABSORPTION OF BILE

The bile from the liver reaches the intra-acinous bile capillaries and flows by way of the interlobular bile-ducts into the hepatic duct From there it reaches the gall-bladder by way of the cystic duct and the duodenum by way of the common duct. With a fasting stomach the sphincter of the common duct is closed This sphincter opens by a pressure of 700 mm water or, better soda solution The secretory pressure of the bile is only 200 mm water The bile cannot overcome this resistance and enters the gall-bladder by way of the cystic duct, where it accumulates until the next meal As soon as the digestive products leave the stomach and reach the duodenal mucous membrane in the region of the papilla Vateri a rich flow of bile enters the duodenum through relaxation of the sphincter of Oddi The digestive products of protein have the greatest influence on the production of bile Some of the bilirubin is reabsorbed, as is shown by the small quantity of that substance normally contained in the blood-serum According to the colorimetric determination of Hymann van den Bergh¹⁴ it is 1 to 400,000 or 1 to 250,000 the threshold being 150,000 Especially rich in bilirubin is the blood in the umbilical cord of the newborn The threshold of the bilirubin in the blood varies in different animals and is lowest in the dog

Functions of Bile —H G Roger¹⁵ enumerates the functions of bile in the following manner (1) Bile has no digestive ferments, but it exercises a zymosthenic influence in the intestine. i e , it increases the action of certain ferments (pancreatic amylase, intestinal lactase, and lipase) (2) It possesses the property of taking up from the epithelium of the intestinal mucosa elaborated ferments, especially invertin, thereby playing an important rôle in the digestion of certain sugars (3) It helps in the digestion and absorption of fats (4) It exercises a special action on albumoses and peptones (5) It prevents the coagula-

tion of mucus by the intestinal mucinase (6) It prevents intestinal putrefaction by favoring the development of certain microbes to the detriment of anaerobic microbes which are mainly putrefactive, it does this by diminishing the secretion of bacterial ferments and by preventing the action of bacterial ferments on fermentable substances (7) It diminishes the action of toxic products, the result of intestinal bacteria (8) It contributes to the nutrition of certain tissues, its suppression causes marked bony changes

The influence of bile on digestion and absorption of fat is utilized by Brulé in differentiating complete from incomplete hindrance to the flow of bile to the intestines If a quantity of fat in the form of sweet cream is given to a patient suffering from icterus and the serum is examined by the dark-field illumination one-half hour later, and no fat globules are found, it indicates complete obstruction to the flow of bile to the intestines

The pathologic changes leading to icterus may occur in one of three places (a) In the ducts of the liver, (b) in the liver cells, (c) in the place (reticulo-endothelial system) where the substances which enter into the formation of bile are formed

Accordingly, we speak of mechanical icterus caused by the obstruction to the flow of bile in the biliary ducts (icterus due to stagnation of bile), dynamic icterus, the result of functional disturbance in the liver cells (diffusion icterus, icterus by parapneumonia—Minkowski), hemolytic icterus, the result of excessive destruction of red blood-cells in the spleen

As useful as this division is from the clinical standpoint, it must be remembered that there are mixed forms Primary hemolytic icterus may lead to parenchymatous changes in the liver and formation of bile thrombi in the ducts, thereby causing an icterus of mechanical, dynamic, and hemolytic nature Primary disease of the larger and, especially, the smaller ducts may cause secondary changes in the liver cells Primary changes in the liver cells may compress the ducts, leading to obstruction, or they may cause obliteration of the ducts by thick bile and by bile thrombi

Before entering into a clinical discussion of the most important diseases accompanied by icterus, it is advisable to adhere to the teachings of Naunyn, to first outline briefly the main manifestations caused by all forms of icterus irrespective of their cause. These manifestations are

1 Yellow discoloration and the presence of bile pigments in the tissues. The yellow discoloration is the most striking symptom. It may vary from the yellow color of fresh sulphur to that of canary or orange yellow. After a while the skin may become greenish, olive green, and in protracted cases greenish gray. The nerve and muscular tissues remain unchanged.

2 Bile pigments and other biliary constituents in the urine, secretions, exudates, and transudates. The urine contains an excess of bile and bile acids and often yellowish hyaline casts, the so-called Nothnagel casts. In addition to the bilirubin there is often also urobilin unless the common duct is entirely closed. In many cases there are also bile acids in the urine.

3 Intestinal secretion is poor in bile. In complete obstructive jaundice the stool is persistently acholic unless a diet rich in meat is taken, in which case the blood-pigments in the meat may lend color to the stool. On the other hand, it must be remembered that clay-colored stool may be bile containing if the bilirubin is completely reduced to urobilin. Bile-free stool has a cadaveric odor. Microscopically it shows fatty acid crystals if it is due to obstruction of the ducts, and if pancreatic digestion is not interfered with. If the obstruction is the result of cancer at the head of the pancreas, the fat digestion is entirely interfered with and fat globules are found in the stool. The stool is then of a buttery character with a glistening surface. The more complete the obstruction the greater the quantity of fat in the stool. At times as much as 80 to 85 per cent. of the fat ingested may be found in the stool.

4 Metabolic and nutritive disturbances are evidenced by the marked loss of weight and great weakness. In acute yellow atrophy of the liver there is also disturbance in protein metabolism, as shown by the presence of leucin and tyrosin in the urine.

5 Anomalies in the blood and circulatory system. The

anomalies in the blood are manifested by secondary anemia, and in the infectious and suppurative types by leukocytosis. In hemolytic icterus the blood changes are characterized by the diminished resistance of the red blood-cells to hypotonic sodium chlorid solution, by an increase in reticulated red blood-cells, and by the presence of mononucleosis. In the blood-serum the bilirubin is increased in all forms of icterus. The indirect bilirubin is increased in hemolytic icterus. The bile acids are also increased in all forms of icterus with the exception of the hemolytic form. This explains the absence of itching in that form of icterus.

6 Anomalies of the nervous system are characterized by irritability and in more severe cases by cholemia, delirium, and convulsions.

7 Anomalies in the liver and spleen. The liver is enlarged and tender, which is mostly due to cholangitis and hepatitis. In many forms of icterus there is also enlargement of the spleen. The enlargement of the liver and spleen does not depend upon the gravity of the disease, as in the most serious form, namely, that due to carcinoma of the pancreas, in acute yellow atrophy of the liver the liver is only moderately enlarged, and the spleen not at all.

CLINICAL SYMPTOMS ACCORDING TO THE SEAT OF THE DISEASE

Obstructive Form—The most common cause of icterus is obstruction of the bile-ducts brought about most frequently by calculi in the common duct.

This form of icterus, as is well known, is most readily recognized because it is preceded, in the majority of cases, by a severe attack of cholelithiasis. The time of the occurrence of the icterus after the attack and the severity of the same depend upon the degree of obstruction. If the obstruction is caused by a small calculus which can either pass into the duodenum or back into the cystic duct and from there into the gall-bladder, or if caused by associated spasm of the sphincter, the icterus is very mild and transient. A moderate yellow discoloration of the scleræ and possibly of the skin, and a bile-containing urine may be the only indication of the existence of an obstruction.

The icterus subsides with the relaxation of the spasm, after which the stone passes the sphincter. This form is also clinically characterized by tenderness over the liver or gall-bladder region. There is often spontaneous pain in the right shoulder and almost invariably tenderness over the right shoulder-joint. The liver is but moderately enlarged. The gall-bladder is, as a rule, not enlarged or but slightly so.

A more severe form of icterus due to a stone lodging in the common duct or to superadded inflammation of the duct, may begin with very severe chills and elevation of temperature as high as 104° and 105° F (Charcot's fever). A moderate leukocytosis may also be present with considerable enlargement of the liver, especially of Riedel's lobe, which may be so marked as to simulate a large gall-bladder. The temperature, however, does not persist very long, it subsides after two to three days. The jaundice may deepen and be so persistent as to necessitate immediate operation in order to avert the possibility of cholemia. The deepening of the icterus, however, does not always necessitate immediate operation. At times, the jaundice may even last a few weeks and then gradually subside. If the icterus does not subside within a few days and the liver remains large and painful, it is most likely due to infection of the smaller ducts. Such an infection does not preclude complete recovery without surgical interference. Obstruction of the large ducts may at times be caused by intestinal worms.

Icterus caused by carcinoma of the gall-bladder or ducts deserves special consideration. If the gall-bladder alone is involved, the icterus may be absent for a long time and the only symptoms present would be pain over the right hypochondrium and enlargement of the gall-bladder. In most cases, however, the smaller ducts also become involved. It would be reasonable to expect that icterus would become very marked early in the disease. This is, however, not the case. For many months there may only be a moderate discoloration of the scleræ, an increase of bile pigments in the blood, enlargement of the liver, and hardly any discoloration of the skin. This is the reason why this disease is so frequently overlooked and diagnosed only

during the very late stage. Even during the terminal stage of the disease, icterus is only of moderate degree and itching is seldom present. This is due to the fact that the ducts probably dilate and allow the bile to pass into the intestines.

Obstructive icterus may be caused by pressure on the large ducts from without as, for instance, tumors (lymphosarcoma), tuberculous and inflammatory glands, also by periduodenal adhesions, ptosis of the liver with kinking of the gall-bladder, and aneurysm of the hepatic artery and aneurysm of the abdominal aorta. Even aneurysm of the superior mesenteric artery may compress the common bile-duct. Cases have been reported where perforation of a hydatid cyst of the liver into the common duct caused obstructive jaundice. Pressure may be caused by an inflammatory tumor of the pylorus secondary to a stenosing ulcer, or by an inflammatory tumor of the head of the pancreas, the so-called Riedel's tumor.

Carcinoma of the head of the pancreas obstructing the papilla Vateri is a very important factor in the causation of icterus. This form starts insidiously, but develops very rapidly. The icterus deepens and within a short time the entire skin and scleræ become greenish or greenish gray. At times the patient has the appearance of a mulatto. Emaciation and weakness develop very rapidly. The obstruction in these cases is complete, as can be demonstrated by the absolute absence of urobilin in the urine and the absence of bile in the stool and duodenal contents. The pancreatic ferments are absent in the stool and duodenal contents, especially the lipase and trypsin. The liver is but moderately enlarged but the gall-bladder is very much enlarged. The law of Courvoisier, that a very large gall-bladder with icterus spells carcinoma of the head of the pancreas, is unusually valuable. There is marked resistance in the upper quadrant of the abdomen and in emaciated people the tumor of the pancreas can be easily palpated. Sugar in the urine is, as a rule, absent. This was pointed out by Neusser and attributed to the fact that in carcinoma of the pancreas, both the external and internal secretions are diminished or absent. The clinical observation that the absence of sugar in the urine in

obstructive icterus favors the diagnosis of carcinoma of the head of the pancreas, holds good in the majority of cases. The explanation for it, however, is not that of Neusser. The work of Banting, Best, and their associates in the Toronto School explains why there is no glycosuria in cancer of the pancreas. Cancer affects the acini of the pancreas and completely obstructs the pancreatic ducts. The islands of Langerhans are unaffected. Hence, the internal secretion is even increased. Cancer of the pancreas almost corresponds to the ingenious method of Banting and his co-workers in the experimental production of insulin. There is in reality a degeneration of the glands of external secretion in cancer as is produced in dogs by the Toronto workers. The absence of sugar in icterus associated with cancer of the pancreas is emphasized because in interstitial pancreatitis of luetic origin, which may also be associated with icterus, sugar is present in the urine, owing to the fact that in this affection the islands of Langerhans are involved.

The itching in obstructive jaundice, especially that due to complete obstruction, is unusually severe and is most marked at night. In the benign type of obstruction of the larger ducts not associated with severe infection the pulse is usually slow. In the malignant type of obstruction the pulse is usually rapid—another important differentiating sign pointed out by Neusser.

Disease causing obstruction of the larger ducts, if persisting for any length of time may spread to the smaller and even smallest bile-ducts, especially is this true if the obstruction of the large ducts is caused by an infection. Large stones in the ducts seldom if ever lead to involvement of the smaller ducts, but if the larger ducts are infected by colon, typhoid, or paratyphoid bacilli, and as in very rare cases, by streptococci, the infection frequently spreads to the smaller and even the smallest ducts, causing cholangitis and cholangiolitis, respectively. In such cases the liver may become very large, tender, and the icterus persist and even become chronic.

Severe infectious cholangitis is most often of hematogenous origin and due to staphylococci, colon bacilli, more rarely typhoid and paratyphoid organisms. In addition to the icterus there

are chills, temperature, and severe pain over the liver. If supuration develops, it leads to multiple abscesses. There is a very marked leukocytosis. It is self-understood that in such cases the hepatic cells are also affected.

Cholangitis and cholangiolitis with chronic icterus and enlargement of the spleen may run a course of several years. The disease is characterized by intermissions during which time the icterus is less marked and the liver and spleen less enlarged. During the period of active symptoms, on the other hand, there is, as a rule, hepatic colic, the liver and spleen become more enlarged, and jaundice deepens. There is also leukocytosis (20,000 to 30,000) and a polynucleosis. The disease is often wrongly interpreted as Hanot's cirrhosis. It is, however, entirely different, as Hanot's cirrhosis affects primarily the liver cells and only secondarily, if ever, the ducts, chronic cholangiolitis affects the duct primarily.

Somewhat rarer than the calculous obstruction of the large ducts in the causation of icterus is the non-calculous obstruction first pointed out by Naunyn.¹⁶ The gall-bladder is the most frequent seat of the disease. Chronic cholecystitis of non-calculous nature is a very frequent occurrence. Chronic affections of the gall-bladder are not necessarily always of bacterial origin. Rovsing,¹⁷ who recently studied the diseased gall-bladders of cases operated upon in his clinic bacteriologically, arrived at the conclusion that in about 60 per cent of the cases no bacteria were found. These studies are no proof that the bacteria were not the primary cause, and that the diseased areas became bacteria free after the infection had become chronic. Such conditions are encountered in other organs of the body as the pleura, peritoneum, endocardium, etc. Whether of bacterial origin or not, chronic disease of the gall-bladder may lead to infection of the larger and even the smaller ducts.

A special form of chronic infection of the gall-bladder causing secondary changes in the ducts and even in the liver cells and leading to chronic icterus, deserves special mention, namely, the one described by Schottmuller and attributed to the *Streptococcus viridans*. Schottmuller named this infection *cholecystitis lenta*.

Non-calculous cholangitis from whatever cause is differentiated from the calculous type by the fact that the attacks of pain are not as severe, the jaundice is not as deep, the stool is only moderately acholic, and the urine contains only small quantities of bilirubin, urobilin, and bile acids. In cholecystitis lenta, secondary anemia and enlargement of the spleen are also present.

Dynamic Form—The dynamic form of icterus is caused by primary disease of the cells of the liver. It may be brought about by a great number of causes and its severity varies according to the underlying factor.

The most common form of icterus due to disease of the liver cells is the so-called catarrhal icterus. This form deserves special discussion. It is well known that Virchow attributed this form of icterus to the formation of a mucous plug (*Schleimpfropfen*) in the duodenum obstructing the sphincter of Oddi, the plug of mucus being caused by a catarrhal duodenitis. This theory, although not based on pathologic observations, was for a long time upheld because Virchow was the originator of it, and even today, a man like Herxheimer states that the mucous plug theory ought not be considered a myth. Naunyn deviated slightly from the theory of Virchow. He attributed the disease to a catarrhal duodenitis which, however, does not cause a mucous plug, but a distinct swelling of the duodenal mucous membrane at a point of the sphincter of Oddi which results in obstruction to the flow of bile. Both these theories have no pathologic bases and are insufficient for the explanation of the jaundice. The conception of Eppinger, which in a modified degree was already held by Minkowski, is much more reasonable. Eppinger believes that catarrhal icterus is due to parenchymatous changes in the liver cells which cause hypersecretion of bile with inability of the bile-cells to direct the flow to the ducts. According to Minkowski, the bile escapes through the cellular membrane into the bile capillary spaces and from there into the lymph spaces, and thence into the blood (icterus by parapidesis, Minkowski). According to Eppinger and Umber, the bile capillaries rupture and allow the escape of bile into the blood. During

the World War, Eppinger had occasion to examine four livers of such cases and he found no disease of the duodenum no bile thrombi in the capillaries, but definite changes in the parenchyma of the liver cells. He states that the disease is a milder form of acute yellow atrophy of the liver cells and that in some cases it leads to subacute yellow atrophy with fatal termination. Although it is true that occasionally catarrhal icterus terminates fatally, there is no reason to assume that it bears resemblance to acute yellow atrophy of the liver. It is much more plausible that the icterus which terminates fatally was originally yellow atrophy and wrongly diagnosed as catarrhal icterus. On the other hand, it must be conceded that the disease constitutes a mild affection of the epithelial cells of the liver which is of a toxic nature and may undergo complete repair. This condition may be favorably compared with the mild nephrosis in the course of acute infectious disease in which *restitutio ad integrum* takes place. It is interesting to note that Westphal found *Lambia intestinalis* in the duodenal contents in 4 out of 18 cases of catarrhal icterus. Whether these intestinal worms bear any causative relation to catarrhal icterus is very questionable.

The disease is clinically characterized by prodromal gastrointestinal symptoms, not due to gastro-duodenitis, but to disturbance in the biliary function. After a few days of prodromal symptoms, consisting of loss of appetite, bad taste in the mouth, fulness in the epigastric region, and marked constipation, the scleræ first become icteric and then the icterus gradually spreads to the rest of the body. The skin is canary yellow, but if it lasts longer it may even become greenish or greenish gray. The pulse is very slow and asthenia is quite marked. At times the loss of weight may be considerable, although this is not the rule. The urine may at first contain only urobilin and no bilirubin and later it may contain bilirubin and no bile acids. In other words, it has somewhat the characteristics of the French dissociation icterus. The stool is intermittently acholic. There may be a bile-colored stool, showing that the obstruction is not complete. The intermittent obstruction is most likely due either to the concentrated thick bile in the ducts or to compression of

the ducts by enlarged cells. The liver is usually enlarged but not tender. The spleen is never enlarged. The gall-bladder may be intermittently enlarged. The fact that the liver cells are diseased is also proved by the positive galactose test. It must be strongly emphasized that this form of icterus never occurs in epidemics and never affects members of the same family. This differentiates it from other infectious types of icterus to be mentioned below.

Catarrhal icterus lasts from three to six weeks, sometimes eight weeks. Cases have been described where it lasted as long as eight months. In the protracted cases, it is usually accompanied by chronic cholangitis and cholangiolitis, by enlargement of the liver and spleen, and by leukocytosis.

Infectious icterus occurring in epidemics is exemplified best by that form which has recently been described by Blummer¹⁸. Infectious icterus may also be caused by paratyphoid bacilli and may run a course similar to epidemic icterus. The diagnosis of this form can only be established by the paratyphoid agglutination test. In neither form is there complete obstruction to the bile. Often there is leukocytosis. The disease lasts from one to two weeks and terminates favorably. Infectious jaundice described by Blummer shows wide variations as regards the type which the outbreaks may assume. (1) The family outbreak, (2) the institutional outbreak, (3) the city outbreak, (4) the country outbreak, (5) the state outbreak. About 72 per cent occur during the fall and winter and the remainder during the other periods. It is a disease of childhood and adolescence and it is transmitted by personal contact. The incubation period is usually about seven to ten days, the shortest is two days and the longest eight days. The onset is usually sudden, with nausea, vomiting, constipation, severe headache pain in the back and limbs, epigastric pain, and fever. On the fourth and fifth days the fever disappears and jaundice appears. Sometimes the jaundice may be delayed as long as twenty days. It varies in intensity from a mild yellow to almost a bronze tint. It persists from one week to ten days, but may disappear after a few hours and rarely does it persist for six weeks or even longer.

Itching is rare The pulse is rapid The liver is enlarged The spleen is sometimes enlarged There is usually a leukocytosis from 16,000 to 24,000 The prognosis is good

Another very important form of infectious jaundice due to primary disease of the liver cell is represented by so-called Weil's disease in which, in addition to the high temperature, enlargement of the liver and spleen, and jaundice, there is nephritis and marked diarrhea A very important diagnostic measure in Weil's disease must be mentioned If about 15 c c of blood is taken from a patient suffering from Weil's disease and injected into the heart of a guinea-pig, the liver of that guinea-pig will be found to contain spirochetes on the third day after the injection

Spirochetosis icterohæmorrhagica was described in 1916 by Inada, Ido, Kaneko, and Ito, in Japan Noguchi showed that it is analogous to Weil's disease

Icterus caused by functional or true parenchymatous changes in the liver cells occurs in the course of severe infectious diseases It is encountered in grave forms of typhoid fever, typhus, and in severe pneumonia In lobar pneumonia it may be present if the right lower lobe is affected, not necessarily influencing the prognosis

The icterus in infectious diseases and, particularly, in pneumonia has been considered by some authors to be of hemolytic origin, either the result of hemolysis of bacterial nature, or, as in pneumonia, the result of extensive involvement of the lungs There is, however, no reason to accept such an assertion Infections causing icterus by hemolysis are usually of chronic nature That extensive inflammatory involvement of lung does not cause icterus is proved by the fact that, although massive pneumonia is such a frequent occurrence, icterus complicating pneumonia is very rare It is, therefore, no doubt that the icterus is due to the toxic affection of the liver cells

Chronic icterus with large liver and spleen, lasting for years and terminating fatally, may at times occur without any etiological factor and, therefore, must be attributed to progressive catarrhal icterus Such cases were carefully studied by Eppinger and he attributed them to chronic yellow atrophy of the liver

Icterus gravis is a special form of severe jaundice caused by degenerative or autolytic changes in the liver cells giving rise to severe hepatic insufficiency. It may be due to a number of causes, among which are spirochetosis ictero-hæmorrhagica, yellow fever, and diffuse hepatitis of staphylococcic or streptococcic origin. It is at times encountered in pregnancy. Icterus due to arsenic, phosphorus, or mushroom poisoning is also of dynamic origin.

A special form of icterus gravis is acute yellow atrophy of the liver. This affection was unusually rare up to the time of the recent war. After the World War it became quite frequent in Central Europe. Umber,¹⁹ who carefully studied this condition, arrives at the conclusion that the disease is due to an impoverishment of glycogen in the liver cells. The presence of glycogen in the liver undoubtedly is a protective agent for the liver cells. Detoxication, as well as the power of assimilation, which are synthetic functions of the liver cells, can only continue if the liver cells contain glycogen. If the glycogen disappears from the liver the liver cells lose their protective property against hepatrophic noxious substances proportionally to the amount of glycogen lost. During the World War inadequate and improper food led to the impoverishment of glycogen in the liver, thereby exposing the liver cells to autolytic parenchymatous changes. Kimura,²⁰ in the Institute of Marchang examined a liver in a case of subacute yellow atrophy two hours after death and he could not find any glycogen in the liver cells. In two of Umber's cases the late Versé also found a marked diminution in the glycogen of the liver cells.

The jaundice during the early stage of syphilis and that due to salvarsan may be caused by acute yellow atrophy of the liver. The symptoms are grouped in two stages. The first stage is characterized by malaise and other symptoms of catarrhal icterus which last for five to six days, rarely a few weeks. The second stage is characterized by grave nervous symptoms and general toxemia, severe headache, restlessness, twitching delirium, convulsions, transient paralysis, squint, dilated pupils, and persistent vomiting. The skin may show hemorrhages and

there may be blood in the vomitus, feces, and urine. Epistaxis and menorrhagia may occur. In the beginning the liver may be enlarged, but it soon recedes. The urine is diminished and contains bilirubin, urobilin, bile casts, and leucin and tyrosin crystals. It may terminate fatally after a few days or after a few weeks. In some cases jaundice persists for months. Rolleston²¹ reported a case where the disease lasted two years. In these cases there is a nodular hyperplasia of the liver. In some chronic cases the clinical features are those of rapidly progressing cirrhosis of the liver with ascites.

The prognosis is fatal in the great majority of cases. There are, however, a few cases on record in which recovery took place.

The icterus caused by phosphorus poisoning is also due to yellow atrophy.

Hanot's cirrhosis, as is well known, is characterized by chronic jaundice, periodic attacks of abdominal pain and fever, enlargement of the liver and spleen, absence of ascites, and by its preference for the young. It is a very rare disease. It was originally thought to be due to an ascending infection of the bile-ducts from the duodenum. This has been disproved. The icterus is not an obstructive one. The pathologic changes are confined chiefly to the liver cells with a great deal of fatty degeneration. There is at times secondary involvement of the ducts.

Eppinger, who observed 8 cases clinically for a number of years, 6 of which came to autopsy, states definitely that the icterus is certainly not due to obstruction. Clinically, it has some of the characteristics of the hemolytic type. In one of Eppinger's cases the removal of the spleen had a very favorable influence on the disease. At autopsy the liver, in his cases, showed bile thrombi in the bile-ducts, dilated and ruptured bile capillaries, and marked deposits of iron in the Kupffer stellate cells—in other words, the same characteristics as are encountered in hemolytic icterus. The spleen did not show the same changes as in hemolytic icterus. On the basis of that experience, Eppinger states that the spleen in hypertrophic cirrhosis of the

liver bears intimate relation to the jaundice and that the icterus in hypertrophic cirrhosis of the liver is of hemolytic and most likely of splenic origin. He definitely points out, however, that the primary disease lies in the destruction of the liver parenchyma. The alimentary canal is frequently inflamed. The pancreas may at times show fibrosis. It is a very slowly progressing disease. Malaise with vague abdominal symptoms and pruritus with enlargement of the liver may precede the jaundice a number of weeks. The enlarged and painful liver may for a long time be the only symptom leading to exploratory laparotomy. The jaundice is at first slight, becoming later dark greenish. Itching is likewise very severe. Occasionally the lymphatic glands in the axilla and groins are considerably enlarged. Death usually results from toxemia. At times the rapid degeneration of the liver cells may cause symptoms of acute yellow atrophy. Intercurrent infections like erysipelas, pneumonia, or even peritonitis are not uncommon. At times the disease does not last longer than two years.

Obstructive biliary cirrhosis consisting of fibrosis of the small ducts may sometimes be the cause of chronic jaundice. The causes for this condition, according to Rolleston, may be infective cholangitis with pericholangitic cirrhosis. The clinical picture of this disease is much like that of Hanot's cirrhosis.

Carcinoma of the liver causes jaundice if the organ is the primary seat of the disease. Metastatic carcinoma of the liver usually causes ascites.

It must again be emphasized that icterus, the result of primary disease of the liver cells, causes bile thrombi in the capillary ducts and secondary cholangitis in the great majority of cases. Brauer rightfully termed these thrombi the casts of bile capillaries analogous to the casts in the tubules of the kidney in cases of nephrosis or nephritis.

It is also important to remember that in icterus gravis the bile-ducts may contain white bile, showing that the function of the liver cell is practically destroyed. In these cases the fact that the jaundice persists shows again the extrahepatic production of bile pigments.

HEMOLYTIC ICTERUS

This disease is of great clinical importance and has given rise to considerable discussion. Naunyn and his pupils even today adhere to the teachings that hemolytic icterus is usually an obstructive icterus, the primary process being hemolysis. The excessive hemolysis causes pleochrome and highly concentrated bile in the liver cell which disturbs the function of the liver cell proper, also bringing about thrombi in the bile capillaries and so leads to obstruction. According to Naunyn, hemolysis may be of an exogenous origin if caused by poisons like toluylendi-amin, arseniuretted hydrogen, and aniline dyes. These poisons destroy the red blood-cells and the hemoglobin is converted into bilirubin. Some poisons may cause free hemoglobin to be converted into methemoglobin.

The second group is of endogenous origin, as exemplified by transfusion, hemolysis, and paroxysmal hemoglobinuria.

Toxic hemolytic icterus is best studied in cases of toluylendi-amin and phosphorus poisoning.

The clinical picture of hemolytic icterus, also known as chronic splenomegalic hemolytic jaundice, was already known to Morgagni, who named it suppression icterus. In 1900 Minkowski gave the first clinical description of the disease. He described 8 cases occurring in the same family during three generations, in whom congenital icterus had lasted throughout life, together with urobilinuria and large spleen. This peculiar affection did not shorten life. Some later reports by Chauffard, Widal, and Abram followed from the French school. The congenital form is named the type of Minkowski-Chauffard-Widal. The acquired form is named the type of Hayem after the author who first described it. In both types the icterus is not very deep and itching is never present. There is only an increase of indirect bilirubin and the presence of urobilin in the blood, but there are no bile acids in the urine. There is a diminished resistance to hemolysis of the red blood-cells when exposed to hypotonic salt solutions as pointed out by Chauffard. Normally hemolysis begins in 0.42 per cent sodium chloride solution, whereas in hemolytic jaundice the corresponding limits are

0.6 and 0.42 per cent. The red corpuscles are smaller than normal. Many reticulated red cells are found. Widal correctly pointed out that this phenomenon is not present in all cases.

In the acquired form the jaundice is less marked and the anemia is much more prominent than in the congenital form. In both forms, especially in the congenital, attacks of biliary colic occur often. In some cases gall-stones are also found. Both types are accompanied by hemolytic crisis, characterized by marked enlargement of the spleen, which is painful, due to perisplenitis, elevation of the temperature, and marked anemia. The periodic increase in icterus, anemia, and enlargement of the spleen may be brought about by a number of causes, such as cold weather, excessive eating, long marches and, according to Parkes Weber²² and Downes,²³ it may also be brought about by pregnancy and mental shock or worry.

Removal of the spleen in these cases is a curative agent. Icterus associated with pernicious anemia and aplastic anemia belongs to the hemolytic type of icterus.

Icterus associated with heart disease has been the subject of much discussion. Naunyn and Gerhard are of the opinion that it is of obstructive type. Other observers, like Eppinger (loc cit), Lepehne, and A. F. Fishberg,²⁴ are of the opinion that it is of hemolytic origin. It may be stated that moderate icterus or the cyanotic icterus associated with decompensated cardiac disease is, as was recently pointed out by Fishberg, the result of the bile pigment formed in lung infarcts and in transudates.

In cases of cardiac decompensation with tricuspid insufficiency, the icterus is due to disturbance in the liver cells. The symptoms are, in reality, those of liver insufficiency (nausea, vomiting, drowsiness). Such a condition is also often present in thrombosis of the right coronary artery. The liver is markedly enlarged and is very tender in these cases.

Biliary cirrhosis, the result of heart disease, may give rise to icterus, both as the result of changes in the liver cells and obstruction in the biliary ducts. Gall-stones in these cases often form as a result of the marked impediment to the flow of bile. This fact is important to bear in mind in order not to over-

look the presence of gall-stones in cardiac disease. One often encounters suppurative cholecystitis as a result of gall-stones in cardiac disease, necessitating operative interference in spite of the grave condition of the circulatory system. In decompensated heart disease all three forms of jaundice may occur, namely, that due to hemolysis, that due to affection of the liver cells, and that due to obstruction of the biliary ducts.

Rare forms of icterus of transient nature deserve mention. These include icterus, which may come on during the menstrual period, that following tuberculin injection, vaccination, and after the administration of salvarsan and even mercury.

Icterus occurring after psychic trauma, known as icterus ex emotione, is not infrequently encountered. After very severe mental shock icterus suddenly develops. It has been attributed to spasm of the sphincter of Oddi. The spasm theory, however, does not satisfactorily explain this form of icterus. The studies of L. R. Muller and R. Greving²⁵ on the innervation of the liver offers a more satisfactory explanation. R. Greving studied the course of the nerves in three fetal livers and in two adult livers macroscopically and microscopically in order to determine to what extent the sympathetic and vagus nerves are concerned in the innervation of the liver. He found that no fibers from the splanchnic nerves go to the liver. These nerves merge with the celiac plexus and from there fine nerve bundles accompany the hepatic artery, through which they reach the liver. Some fibers from the vagus, on the other hand, enter directly into the liver, especially those of the left vagus. The hepatic artery at its entrance into the liver is surrounded by extensive nerve bundles. The nerve bundles are scarce in the region of the portal vein. The nerve-fibers from the splanchnic and vagus nerves have chiefly vasomotor function and, to a certain degree, they also regulate the metabolic processes in the liver. These experimental studies and those of the Kraus school (Brugsch, Dresel, Loewy, Leschke, and Schneider) showed that the liver does not only exercise its metabolic influence on the material brought to it by the blood-stream, but its work is also under the control of the nervous system, the center, therefore, being in the

midbrain and in the medulla oblongata According to the examination of Eiger the vagus increases the flow of bile and the sympathetic nerve diminishes the flow of bile In the case of emotional icterus it is the overstimulation of the sympathetic nerve which brings about retardation of the flow of bile, resulting in absorption icterus

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CLINIC OF DR H H FELLOWS
CHIEF, GASTRO-INTESTINAL DEPARTMENT ASSISTED BY
DRS WEINTRAUB, RUSSELL,
AND
MISS FRANCES EVANS
CLINIC EXECUTIVE

CORNELL CLINIC, CORNELL UNIVERSITY MEDICAL COLLEGE, TO
STUDENTS OF FOURTH YEAR CLASS

EXAMINATION OF PATIENTS WITH GASTRO-
INTESTINAL SYMPTOMS

DR. FELLOWS You have had your lectures and recitations on the symptomatology and diagnosis of gastro-intestinal conditions, and before you start the more practical part of the work I wish to explain to you the system we are following in the clinic and in private practice

You are beginning the practical examination of ambulatory patients complaining of gastro-intestinal symptoms and it is now necessary to put into practice your theoretic knowledge. The methods which we use and the system which we follow are the result of wide experience and are the ones which we find give us the highest percentage of correct diagnoses.

In the first place, every patient must have a general examination, consisting of a careful history and a complete physical examination. This, plus the functional and laboratory tests, gives us our diagnosis.

History taking, I presume, you are familiar with, and yet, perhaps, it is not a waste of time to run over the way in which we do it. It is best to take a chronological history, inquiring into the family history first, where we often acquire facts such

as a tendency toward tuberculosis, mental instability, and occasionally a suspicion of luetic inheritance. Personal history should begin with childhood and not too much attention paid to the common diseases of childhood, as they are usually not significant, unless leaving permanent scars or residuæ. The adolescent period is often illuminative, as there we may learn of early attacks of so-called "indigestion"—nausea and vomiting. Occasionally early symptoms of ulcer are obtained. If there are early and persistent symptoms, one must be on guard against congenital adhesions or abnormalities. In female patients a menstrual history is included in the adolescent period. Coming to the adult life, inquire separately as to each system, the circulatory, respiratory, and genito-urinary—here especially for venereal infection—nervous system, surgical history, and the weight over a period of years. What we are trying to do is to properly evaluate any of the conditions which may give rise to gastro-intestinal symptoms, and yet be due to other than gastro-intestinal lesions. Following the general history, ascertain in detail, if possible, symptoms referable to the gastro-intestinal tract.

The blanks which I have handed to you give the headings of the gastro-intestinal history, and include chief complaint, duration, onset, course, appetite, deglutition, pain, regurgitation, nausea, vomiting, vomitus, pressure, distention, belching, food intolerance, jaundice, chills, fever, "acute attacks," bowel function, feces, mucus, blood, diarrhea, hemorrhoids, and kidney symptoms. I shall just run briefly over some of the main items and leave it for you to elaborate upon them. For instance, the onset and course of the condition is usually best elicited as a sort of a story told by the patient, beginning with the first symptoms referable to the gastro-intestinal tract. The history follows through the period of time from which symptoms were first noted to the present. A story form of history gives a very good idea of the course of the disease, but, after taking this type of history, one has to ask routine questions to be sure not to leave out important facts. Regarding the appetite and deglutition, it is not necessary to say more than that

one should be on guard when questioning with regard to the swallowing of food, to be sure to eliminate or gain information which would lead one to suspect cardiospasm or carcinoma of the esophagus. When we question in regard to pain, there are several factors which must be considered—character, time of appearance, radiation, whether with the pain there is also tenderness in the same locality, the relation to food, and what measures, if any, give relief. If there has been more than an occasional vomiting attack, we want to know what seems to bring it on, whether the food comes up easily, as though regurgitated, or whether the vomiting is projectile in character. It is also important to note the type of food vomited, whether there is blood present, whether the food is that taken some time before, which would make us suspicious of retention. Pressure and belching directly after meals, especially when a large amount of food is not eaten, make one suspicious of either hurried meals or incompletely masticated food. Though bands or adhesions about the duodenum may give rise to these same complaints. If there have been attacks of acute indigestion, one must also be sure to eliminate renal colic, angina pectoris or coronary sclerosis, Dietel's crisis, and, more frequently, gall-stone colic. If gall-bladder involvement is suspected, jaundice, chills, and fever may be present with or following the attack. A careful history of the bowel function is of course, necessary, though many patients are prone to say they are constipated, and let it go at that. We must know a good deal more than that the patient is constipated. We want to know the degree, whether laxatives are habitually used, and, if so, what and how much, whether the constipation alternates with diarrhea, if there has been blood or mucus in the stools. I have not gone into great detail on the way in which the cardinal symptoms are elaborated upon, but I have just given you a hint as to how one questions the patient complaining of gastro-intestinal symptoms. In the history, also inquire well into illnesses, habits, the home environment, family relations. The mental attitude, such as worry over other members of the family or over finances, must be gone into. This, in the office, of course, will be done by each one of you, in

the clinic it is done by Miss Evans, whom I have asked to very briefly outline the points of special interest

MISS EVANS In interviewing the patient after the initial examination it is important to consider not only what *we* wish to get out of the interview but what the patient himself wants to know. As they bear on the physical condition of the patient and the medical plan, we are interested in the patient's life and habits, the environment at home and at work, family relations, his worries, how and where he eats his meals, his daily routine, and financial situation. His interest, however, is subjective. What is wrong with him? What can we do to help him? How much will his examination and continued treatment cost him in time and money? Will the examination hurt? In going out to get the information we want, we must not forget the information he wants. This first interview, therefore, is an exchange of data outlining to the patient the details of examination and treatment and getting in return the information we seek. How well we sell this initial contact with the patient is measured by the future response and returns.

The next important contact with the patient is after diagnosis has been made and treatment outlined. Diets and exercises must then be explained, home situations adjusted, country care planned, hospitalization arranged for, work problems talked over, in other words, the medical plan interpreted and explained in the light of the patient's own environment and daily life. Plans for treatment must often be modified or radically changed because of the patient's mode of living, and often the help of the family must be enlisted and their co-operation gained.

At all subsequent visits, the patient is interviewed, after he sees the doctor. Often this is but a friendly contact, but many times new problems will arise or old problems will need checking up and readjusting.

DR. FELLOWS. Following the history, the physical examination is next performed. Digestive symptoms are so frequently due to such remote lesions as coronary sclerosis, pulmonary tuberculosis, decompensated hearts, lues, renal calculi, that one must be very careful to trace to the source all of the

symptoms complained of. So a complete physical examination is necessary. There are none of you who do not know how to make such an examination in the proper manner, but there are a few points in the abdominal examination which may be helpful. Inspection, percussion, and auscultation do not yield much, neither is one able to feel a great deal in most cases, but if you will try and picture the anatomy and know over what organs you are palpating, it is possible to occasionally gain considerable information. For instance, duodenal ulcer tenderness can usually be elicited over the lesion, and in the short broad type of individual the duodenum may be pretty well hidden under the rib border, but in the thin, ptotic type, the duodenum is easily found just above and to the right of the umbilicus and tenderness is found in that location. If the liver is palpable, one must decide as to whether there is an enlargement or whether the organ is ptosed. An enlarged liver can be percussed higher than normal, invading the thorax as well as being palpable below the rib margin. In thin or elderly patients a prominent or sclerosed abdominal aorta must not be mistaken for intra-abdominal mass or growth. Cold hands on the part of the examiner should be avoided, as they cause immediate contraction of the muscles of the patient. The patient should be instructed to void before examination for a distended bladder is not only uncomfortable and makes palpation difficult, but may actually hide some significant, palpable mass. The examination of a patient in a bathtub filled with warm water renders abdominal examination very easy and very satisfactory, even in the more resistant patients, as there is more complete muscular relaxation. No physical examination is complete without a rectal examination, and this is something that is often overlooked, even by the best-trained physicians. There is nothing simpler than exploring the rectum with the finger, and all of you can, with very little practice, become proficient in the use of the proctoscope as well. You will be gratified and surprised to find how much information you will gain if these two examinations are done routinely.

Modern medicine tends toward a multiplicity of laboratory tests and in examining gastro-intestinal patients one should be

very careful to adhere to the simple and recognized tests. The most illuminating of these is the expression of the Ewald test meal one hour after ingestion. One not only gains help in diagnosis, but in treatment, as well, for from this simple test we are able to learn the acidity of the stomach, the emptying time, presence or absence of blood, and character of digestion. Routine urinalysis should be done, and the Wassermann blood-test at the discretion of the examiner. An estimation of the hemoglobin and red blood-cells is frequently of value, especially where malignancy is suspected, and, when advisable, a stool examination should be done as well.

STUDENT Do you do the Lyon test of biliary drainage and examination of duodenal content?

DR FELLOWS We are not doing the Lyon test at present for several reasons. We are speaking of the examination of ambulatory patients, those who come to the clinic and to the office. The Lyon test takes a good deal of time when it is properly done, the results are still under discussion, and, as I told you, we use only the tests which are of proved value. There are a good many examinations which are occasionally useful, but which had better be done in the patient's home or in a hospital. Among these are the Lyon test, the fractional Ewald, and tests for enzyme activity.

After the history, physical examination, and laboratory examinations are completed, the next step is the x-ray examination, without which our findings are incomplete. Dr Weintraub, will you explain the method of fluoroscopic examination?

DR WEINTRAUB We shall discuss now the routine fluoroscopic examination. The apparatus used is a horizontal and vertical fluoroscopic unit of any standard make. The preparation of the patient is simple. Catharsis is not permitted as we wish to maintain the normal status of stomach and bowel, and on the morning of the examination breakfast and all medication is omitted. The patient, if a male, is stripped to the waist, if a female, removes all the clothing except the underclothes and, during the examination, wears a smock. The patient now stands with the back against the fluoroscope, with the screen in front

First we examine the lungs and look for differences in apical shadows, hilus shadows, calcified lymph-nodes, thickening of the bronchial tree, and any unusual markings. In the aortic arch and the heart we consider the contour, size, and unusual pulsations. The patient is then instructed to breathe deeply, in order to observe the cardiophrenic and costophrenic angles, and movement of the diaphragm.

The examination of the abdomen before the barium mixture is administered may reveal the shadow of the liver or spleen, a tumor mass, sclerotic abdominal aorta, curvature of the spine, the distribution of gas in the bowel, and very rarely gall-stones or renal calculi.

Now the patient is ready for the opaque mixture, which consists of 8 ounces of barium sulphate, $1\frac{1}{2}$ tablespoonful of sweetened condensed milk, 1 tablespoonful of cocoa, and 1 teaspoonful of powdered agar-agar, with water enough to make 1 pint. This is thoroughly mixed and makes a smooth, fairly heavy, palatable drink. The subject is now turned to the right oblique position, which means that his back makes a 45-degree angle with the machine, with the left shoulder nearer the machine, and the right shoulder nearer the operator. It is important to understand this position, and also its opposite, the left oblique position, as we use these terms frequently in fluoroscopy.

The esophagus is best seen in the right oblique position and, as the subject drinks, we note the contour, the normal depressions made by the aortic arch and diaphragm, and especially look for irregularities in outline, and spasms. While the patient is still drinking, it is wise to observe the barium entering the stomach, as in this way lesions of the cardia may be discovered. With the palm of the right hand gently press the column of barium upward and examine the cardiac end of the stomach, noting the curvatures and the surfaces. Then, with both thumbs placed a little distance from each curvature, press alternately to test the pliability of the stomach. The patient is then turned back to the original position: *i. e.*, anteroposterior, and consider the shape of the stomach, the size, tone, position, and the character of the peristalsis. The lesser and greater curvatures are ex-

amined in detail and with the patient in the right anterior oblique position the posterior and anterior surfaces are examined. The body of the stomach is examined in the same manner. Now we are ready to fill up the antrum and the duodenal bulb. It is at this phase that the art and skill of fluoroscopy come in, since about 90 per cent or more of the lesions occur in this region. If we merely observe the stomach emptying by its own activity, we usually see a poorly outlined antrum, and incompletely filled bulb. But with the proper manipulation, the antrum and bulb can be easily filled and leave little doubt as to its nature. With the palm of either hand, press the mass of barium through the pylorus into the duodenum synchronously with the peristaltic waves. The normal antrum is dome-shaped and rounded. Note irregularities in outline due to filling defects, folds of the mucus membrane, bands, or adhesions. Also, observe next the width of the pyloric interval, *i. e.*, the distance between the bulb and the antrum, as well as the position of the pyloric stem. The normal bulb is cone-shaped. Note irregularities in outline due to ulcer, adhesions, unusual fixations, and diverticula. Then comes the rest of the duodenum with its fixations, dilatation, stasis, and reverse peristalsis.

The examination so far has all been done with the patient in the erect position, using the vertical fluoroscope. The next step is to examine in the horizontal position with the patient lying flat on the abdomen, the head pillowed on the left forearm and turned away from the examiner, and the right arm of the patient by the patient's side. This makes it possible to turn the subject from one side to the other very easily and one is always sure that the hands will not come in contact with a high tension wire. In the prone position the cardia is visualized very nicely, as gravity fills the upper end of the stomach. With the patient lying slightly on the right side the antrum is also seen very distinctly and one is also able to determine adhesions about the second and third portion of the duodenum. If an esophageal lesion has been detected, the patient is instructed to drink the barium through a tube and, as the fluid takes longer to pass into the stomach, there is an excellent opportunity to observe a question-

able area for a longer space of time than in the erect position. The first examination is completed at the end of the investigation in the horizontal position. In three hours the patient returns and we note the amount of barium remaining in the stomach and its distribution in the small intestine and colon.

On the following morning the patient returns for the twenty-four-hour examination, having been instructed to refrain from moving the bowels, for the stomach and small intestine are usually empty and the colon is filled. Observe the position, tone, pliability, and stasis. If the appendix is visible, consider whether it is fixed, kinked, beaded, or tender. If a lesion of the colon is suspected, a barium enema is administered and we watch the colon fill under the fluoroscope and thus discover filling defects, adhesions, and spasms. I wish to add a few words concerning our check-up system. Occasionally at the first examination a stomach may be in spasm so that we cannot fill up the antrum and bulb to our satisfaction, or there may be an irregularity of outline the nature of which is not clear, or the history may be strongly suggestive of a lesion and we cannot find it. In such cases the patient returns for a re-examination, usually by a second observer, who is unfamiliar with the case, so that we may have an unbiased opinion, and always by the first observer as well, who at this time is quite familiar with the special area which is to be investigated. A dose of bicarbonate of soda is given before the ingestion of the barium meal, as this frequently makes more satisfactory the examination of the antrum or duodenal bulb, though occasionally a course of atropin treatment is necessary before we can definitely rule out spasm. This second, and even third, examination is essential in accurate fluoroscopy.

I shall close by discussing the danger of fluoroscopy, namely, the x-ray burn. The patient is exposed from five to fifteen minutes. Some of our men have been fluoroscoping two to four hours a day for the past five years and no case of x-ray burn has come to our attention thus far. A good pair of lead-lined gloves is the only precaution which we observe. The most essential point in fluoroscopic work is a sufficiently long examination to

make sure of all points and experienced manipulation of the barium filled organs

STUDENT Do you take x-ray plates, or do you depend entirely upon fluoroscopy?

DR. FELLOWS We depend mainly upon fluoroscopic examination, but the ideal method, of course, is to have films available as well x-Ray plates or films and the fluoroscopic examination supplement each other, but, if only one, equally well carried out, were available, I should much prefer the fluoroscopic examination. The points in favor of x-ray plates are that they are available at any time, there is ample time for leisurely study, and if an operation is necessary the surgeon who is to operate can see the lesion which he is to attack. Balanced against these are the factors of expense, expensive equipment is necessary, a good deal more time has to be devoted to the patient, and the interpretation is usually done by one unfamiliar with the history of the case, which decreases the value.

STUDENT How long does it take to complete the examination of a patient, using all of these methods in examination?

DR. FELLOWS The first examination, including the history, the physical examination, and the fluoroscopic examination of the stomach and duodenum, takes about an hour. The succeeding examinations take about fifteen minutes each, except the final one, when a summary of the case is gone over with the patient. This visit usually takes about half an hour. The total time, then, is between one and a half to two hours, and is divided over three or four days.

STUDENT Have you any gauge as to the accuracy of this system of examination?

DR. FELLOWS Yes. Dr. Russell has just finished a study of the histories of 200 patients whom we have examined in this department and who have come to operation. Nothing can give us a better standard of our work than the operative findings, unless it be an autopsy. I shall ask Dr. Russell to give you the figures on these cases.

DR. RUSSELL In the first place, this is not a selected series. I have analyzed the histories of 200 patients who

were examined by the staff of this department and who have come to operation. We have the operative reports either from the surgeon or from the hospital in these cases, and I have divided the statistics in the following way: First, the accuracy of the clinical examination based on the history and physical examination to determine how near right the first impression is as proved at operation. Second, the x-ray plate diagnosis alone. Third, the examination as carried out, including all of our methods. The clinical diagnoses showed that out of the 200 cases, there was a provisional diagnosis in 111 cases, of which 61 per cent were correct and 39 per cent. were wrong. In 89 cases diagnoses were deferred and, therefore, we cannot state whether these impressions would have been correct or incorrect, but the best a clinical examination would give us would be about 50 per cent correct diagnoses. Of 27 cases of x-ray examination with plates alone, 17, or 64 per cent, were correctly and 10, or 36 per cent, were incorrectly diagnosed, but here I wish to say that this is perhaps not a fair estimation. In some cases there were not a sufficient number of plates taken and the work was done under some handicap. I should think that 75 per cent would be a fair figure for x-ray diagnosis by plate method alone. In the Department diagnoses, using all methods for help, we were correct in 186 cases, or 93 per cent, and incorrect in 14 cases, or 7 per cent., and I feel that we should consistently diagnose correctly about 95 per cent. of our cases. It is more interesting to analyze the mistakes than it is the correct diagnoses. In 10 out of 14 cases incorrectly diagnosed, we reported pathologic findings, and at operation either a much less grade of pathology or none at all was found; in other words, we erred on the side of positive findings rather than negative findings. An analysis of the whole 200 cases shows that 80 per cent of the cases were made up of pathology of the appendix, ulcer of both stomach and duodenum, gall-bladder, and carcinoma of all organs. Five per cent were adhesions and 6 per cent. were negative. In other words, 12 patients in whom we found no pathology were operated for one thing or another and our negative diagnosis confirmed. This is very significant. Ten of the

above cases correctly diagnosed before operation were diagnosed by proctoscopic or rectal examination with or without a barium enema for corroboration. This I mention to emphasize the point that about 5 per cent of our cases, especially when the rectum is involved, can be diagnosed by a rectal or proctoscopic examination.

STUDENT Doesn't it take a good deal more experience than the average doctor possesses to use all of these methods?

DR FELLOWS I'm afraid you have missed the point entirely. It does take special training and a wide experience to correctly interpret fluoroscopic findings, but it is none the less true that, using all methods of diagnoses, 95 per cent of the patients complaining of gastro-intestinal symptoms should be correctly diagnosed, and there should be in each community someone with the requisite skill and experience to do the work for the public and the physicians.

